The New England
Journal of Medicine

Copyright, 1938, by the Massachusetts Medical Society.

VOLUME 219 NOVEMBER 24, 1938 NUMBER 21

HENRY PICKERING BOWDITCH AND THE DEVELOPMENT OF THE HARVARD LABORATORY OF PHYSIOLOGY

FREDERICK W. ELLIS, M.D.

NEWTON CENTRE, MASSACHUSETTS

IN THE year 1929 a great physiological conference was held at the Harvard Medical School. As the writer stood before the magnificent group of buildings and was jostled by the crowd from the four corners of the earth, he was saddened by the thought that there was probably not more than one other member of that International Physiological Conference who could cover with his own recollection the modest beginnings of physiological research in the Harvard Medical School, and its ascent, at first slow, and then rapid, to its present commanding position in the institution. The story of that ascent is an inspiring one, but I fear that most of the scientists who labor in the marble palace now devoted to the pursuit of physiology in the institution where systematic teaching of the science as a university function began in this country are ignorant of that story, which should teach them a useful lesson, and create an appreciation of their good fortune which may not now exist. What is true of Harvard University is true of other institutions in our country. Physiology has come into its own in America in the last fifty years. In no other part of the world has there been such a spectacular change in its status and material resources. The medical profession has finally recognized the truth that physiology is the basis of scientific medicine.

It was in Massachusetts that physiology began its rise in America. We might illustrate that rise with a curve beginning with a time abscissa corresponding with 1871, when Henry Pickering Bowditch, just home from the laboratory of the world's greatest teacher of physiology, began his career as a full-time assistant professor in the Harvard Medical School. Physiology began then to be appreciated in our country as a basic medical science, as well as a subject of academic interest; but that recognition came very slowly, even from the most enlightened medical contemporaries.

The writer well remembers that while he was riding on a railroad train in the early eighties, Reginald H. Fitz, then a professor of pathology, sat down beside him and asked what he was doing at the time. When he was told that this recent graduate of Harvard Medical School was occupied with physiology he expressed the opinion that physiology had done about all that it could for medicine. This opinion came from one in thorough sympathy with science, and from one who was destined to be known soon as a great discoverer in medicine. Fitz thought then, as most well-educated physicians in America thought at the time, that physiology had little real promise for medicine, although he was one of the few medical men of that period who evinced an intelligent interest in the young man's scientific work. But what have physiology and physiological methods not done for medicine since that interview with Fitz on a commuters' train to the North Shore? At that time Fitz's science was an anatomical one; now it is largely a physiological one. Subtract the physiological discoveries that have been appropriated by clinical medicine since that ride with Fitz, and what is left of modern medicine? Bacteriology and the sciences which have been annexed to it are largely physiological branches of biology. Ductless glands were then an enigma, and hormones and vitamins were unknown. Biochemistry was just appearing above the horizon, and physical chemistry, which has become an indispensable tool of physiological and clinical research, had not been named. Clinical research has become applied physiology, and many men in the best modern hospitals are working on what are essentially physiological problems with methods and apparatus borrowed from the physiological laboratory. The scientific aspects of medicine have been turned topsy-turvy, for some of the most important contributions to physiology in recent times have been made by pathologists and clinicians.

A few years ago an old friend of mine, a university professor, was desperately ill from an attack of pernicious anemia, a disease that was uniformly fatal in the elder Fitz's day. He was practically bedridden, and could not cross the room unless supported, and then only at the risk of fainting. About two years later I saw him while he was on a vacation at his son's farm. It was a hot summer day, but he had been seeking physical and mental refreshment by running a mowing-machine in the hayfield. It is unnecessary to state who were responsible for this miraculous change in his condition. Minot and Murphy did this great thing, and back of them was Whipple, who forgot that he was a pathologist, and trespassed on the domain of physiology. It is astonishing how many scientific discoveries of the first magnitude are being made by people who have no business to make them, for they make conspicuous the paucity of the output of those who ought to make them and do not. Insulin and diabetes naturally occur to us in this connection. A young man with slight claim to the title of physiologist, and with a limited experience in medicine, with the able assistance of others makes one of the greatest of medical discoveries, and one that is a physiological discovery.

The few men now living who entered the Harvard Medical School sixty or more years ago have seen a wonderful revolution in medicine, and I, who am of their number, have seen things equally wonderful happen in physiology and to physiology since that time. I became a student in the Harvard Medical School in 1877, and made my acquaintance with Bowditch's attic laboratory on North Grove Street about the time that the present Higginson Professor was six years old. The school was little changed from what it was when Bowditch joined the staff in 1871, and the faculty was nearly the same as it was then. Bowditch was a full professor, but I suspect that the emoluments of his post could be cared for easily. The policy which had been inaugurated in the school, in 1871, by President Eliot was in successful operation. The institution had been pretty thoroughly inoculated with German science, which had begun to take, and had already produced a good deal of a blush. The ambitious graduates of the school no longer flocked to Paris for their professional polish, but Germany and Austria were their objectives, and it was German science and methods that Bowditch brought home with him for his pupils. While Bowditch was a student in Germany and for a generation later, German science overtopped that of the rest of the world, and German physiology attained its high-water mark at that time. President Eliot was a chemist, and was well aware of the merits of German science. He, no doubt, set his trap for Bowditch early; in fact, I think that we may say that Bowditch was predestined for the position which he attained. In those days the best in human values was thought to be located in Boston, or in its contiguous territory, and Bowditch originated in that territory. Although President Eliot was quite capable of making a mistake, a fact which I am unaware that he ever publicly acknowledged, he did the best possible thing in making Bowditch the connecting link between the German laboratories and the Harvard Medical School. Germany was then the acknowledged leader of the world in physiology, and Bowditch became the representative of that leadership in the United States.

By starting our imaginary curve at the year 1871 we do not imply that there had been no notable achievements in physiology in this country prior to that date; but it was then that the science first received definite and intelligent recognition from university authorities, and began to be taught and cultivated in an organized and efficient way. This momentous beginning was due to President Eliot, who was probably the first president of Harvard who had an adequate appreciation of the scientific needs and deficiencies of his institution. The medical school was one of the first departments of the university to which he turned his attention and especial interest, and that interest never flagged. I am of the opinion that the greatest work which he did in the university was done there.

The base line of our imaginary curve is not a zero line. Very creditable work in physiology had been done in this country before Bowditch established his laboratory, but the great significance of that laboratory was due to the fact that it was a research laboratory open to qualified medical scientists who had been bitten by the bug which compels a man to search for new facts in a particular branch of his profession. In addition to his German training Bowditch had another eminent qualification for the position to which he was called, for he belonged to a devoted and intelligent family in easy circumstances that was ready to finance his scientific career. In those days the recompense which one received from following such a career consisted largely in allowing someone else to pay his bills. In honoring Bowditch for his part in the rise of physiology in this country we should include in that honor the family that made it possible for him to accomplish what he did. Now the circumstances under which a physiologist works in the United States are so changed as to seem almost incredible to the older generation that remembers the lean and Spartan years. Science and

money are now in alliance, and to a certain extent are interchangeable. Some people have expressed a wish to "soak the rich," a class which for a generation, and until recently, has been inundating this country with every description of educational, artistic, scientific and charitable institution, until we have become the envy and despair of other nations, but I do not expect to hear any loud denunciation of this class from the physiologists who are now enjoying the comforts of a real home. If we should construct a curve of the rise of physiology in this country during the last fifty years, and another curve of the increase of our national wealth in that period, I think that we should find them to be almost exact replicas of each other.

At the time that Bowditch became a professor of physiology it was a gracious Harvard custom to allow an incumbent of a new chair of science to furnish his apparatus, and so he purchased and brought home from Europe instruments that he knew that he would need. In thus providing himself with facilities for work he had predecessors in the university. President Eliot's first teacher of chemistry, Josiah P. Cooke, was appointed professor of chemistry in 1850, but it was necessary for him to illustrate his lectures with apparatus from a juvenile laboratory which he had established in his father's house, and his first laboratory in Harvard was in a cellar. Formerly when a newly appointed professor of science timidly approached the authorities for a place in which to work, he was led to the entrance of some subterranean locality and was told that he might establish his laboratory there. He was fortunate if he did not need a ladder to get into it, and rubbers after he arrived at his destination. Bowditch needed no rubbers in his first laboratory, for it was situated just below the rafters at the top of the building in which it was located. I have a vivid recollection of that laboratory although I never worked in it, as all my research work with Bowditch was done in the Boylston Street laboratory during the first three years of its existence. The main room of the old laboratory, which was not a large one, had for its principal furniture a table for experiments, a sink, a workbench and foot lathe and a tub for frogs. A smaller room which was shared with Quincy, who had just been appointed an assistant in histology, contained cases for instruments and a few books. This was the laboratory and equipment that Bowditch used for twelve years. I do not think that he had an official assistant for the first five years of that time, but George M. Garland became his assistant in 1877. The appropriations for the physiological laboratory were certainly meager. On one occasion in my undergraduate days I experienced a desire for a frog for some scientific purpose, and I ventured to approach Bowditch for a donation from his store. He very gravely and courteously informed me that the funds at his disposal did not warrant a reckless extravagance of that kind, and then the natural goodness of his heart overcame him and he went to his tub and seized the most diminutive batrachian in it, with which I departed with mingled feelings of exultation and remorse. This little incident illustrates the conditions in the physiological department of Harvard in 1878. Five years later Bowditch moved into his Boylston Street laboratory, which was considered palatial, and was probably the best in America at the time, and which to me was one of the most attractive spots on earth. The physiological curve made a large jump upward at this time, and the gradient has been a very steep one ever since.

During my undergraduate days the medical school was housed in a dingy building at the end of North Grove Street amid decidedly humble surroundings that had probably seen better days. The morgue was near at hand, the jail was not far away, and the appearance of the plain brick structure was that of an impecunious relative of the university to which it belonged. I gazed upon it for the first time with the subdued emotion which the tourist feels when he first sees the Pyramids. The building was then only thirty years old, and was in good company, for the Massachusetts General Hospital was just behind it — an institution that has always been so closely knit to the medical school as to seem to be an integral part of it. The class to which I was added had ninety members, and of these only thirty-five had academic degrees; but this fact should not be considered as evidence that the school was functioning on a low plane. Nearly all the students were on a par so far as essential premedical education was concerned, except that a few of the college men had had general chemistry. For the most part the students with whom I was associated, whether college graduates or not, worked hard and were a credit to Harvard, and to the profession in which they afterward achieved success, and some of them distinction. If anyone believes that medical education in Harvard in 1877 was at a low ebb, he is mistaken; but it must be remembered that scientific medicine had just begun to climb upward then, and that the greater part of it that is now familiar to us was then unknown.

When I entered the medical school there was much there to remind us of the past, and especially of the great discovery with which our institution was so intimately connected. Figuratively speak-

ing the atmosphere of the school was permeated with sulfuric ether. Not many rods away was the amphitheater where Warren demonstrated to the world that Morton's method of annulling consciousness in surgical operations was "no humbug." Another John Collins Warren, the grandson of the first, and a genial friend, scholar and gentleman, was a young instructor in the school. Oliver Wendell Holmes, who coined the word "anesthesia," and who joined the faculty in 1847, a year after the ether demonstration, was the idolized professor of anatomy who lectured from one to two in the afternoon, and could make us forget the pangs of hunger at that unconscionable hour. He became at that time another Orpheus charming the inmates of Hades with his lyre. He could even make osteology delightful by embellishing it with poetry, and interlarding it with anecdote. Our professor of surgery was the distinguished and picturesque Henry J. Bigelow, with a patriarchal beard that made him look like Michelangelo's Moses, who was a survivor of those present at the first ether operation in the adjacent hospital. It was he who first proclaimed the discovery of anesthesia. We had the privilege of hearing him speak of his acquaintance with Morton, and of Morton's frequent and prolonged visits to promote enthusiasm in the subject that engrossed his thoughts. We also had another and agreeable reminder of Morton and his ether in my class in the person of an unassuming and popular young man who was Morton's son.

The discovery of ether anesthesia was the greatest that had been made in practical medicine up to the time that it was announced by Bigelow in the *Boston Medical and Surgical Journal* a few weeks after the memorable operation on October 16, 1846. That message astonished and stimulated the whole surgical world, and it came from the Harvard Medical School. We should not forget that Bigelow was a product of the school, and was soon to become one of its teachers, like his father. John Collins Warren had long been a distinguished professor there. Morton, who administered the ether, was then studying in the school, and Charles T. Jackson, who is said to have suggested ether to him, was a graduate. It was the prestige and authority of the Massachusetts General Hospital, which was manned largely by the officials of the Harvard Medical School, that gave ether anesthesia to the world. This is the truth to remember in all debates on the priority of the discovery. That discovery was a physiological one, and it also became of the greatest service in physiological experimentation.

When I began my course in the medical school Bowditch was a stalwart young man of thirty-seven, who seemed to be the embodiment of health and energy, and walked with an eager, springing step, as if he were anxious to get somewhere and had no time to lose. The instruction which he gave in the school was by lectures illustrated with excellent demonstrations, and by quizzes and conferences. A number of the students were given subjects for study in the available literature, and then reported their findings at meetings in which they were discussed. This was an excellent way for broadening and deepening the information which they obtained from the lectures. The course in physiology in the Harvard Medical School was a complete refutation of the view that real instruction in science cannot be given to students without individual work by them in a laboratory. I have less hesitation in saying this because I have been an incurable addict to laboratories of various kinds for more than sixty years. The elementary laboratory is desirable and useful, but its results depend largely upon the kind of student who enjoys its advantages. It is the student who enjoys his work there, and leaves it with reluctance, that derives a benefit from it commensurate with its cost. Knowledge obtained in the laboratory may leave as easily as it came. However, our class did have an infinitesimal amount of practical instruction in the attic laboratory. We were divided into squads, which were summoned to the laboratory twice during the first year. I remember that on the first occasion that I enjoyed this privilege Garland showed us how to make the classic nerve-muscle preparation from the frog's leg, and that on my second visit Bowditch was on hand to initiate us into the mysteries of graphic registration on the smoked drum. This was the tiny germ from which the present luxurious growth of laboratory instruction in physiology in the Harvard Medical School was derived.

While I was an undergraduate student Bowditch did considerable research work in co-operation with his assistant, Garland, or an advanced student or outsider. He also contributed independently a few notes or articles to the English *Journal of Physiology*. During the first twelve years of Bowditch's experience in the faculty of the medical school his work was a grind that he no doubt enjoyed, but all the while he was dreaming of better things, which came with startling swiftness after they once started. After he moved into the new building on Boylston Street a new era began, and his dreams came true.

During my three years' absence from the medical school, after I graduated, I had no very direct knowledge of what was transpiring in its physiological laboratory, but I have no reason to think

that the investigations carried on there during this period were numerous. The English *Journal of Physiology* was established in 1878, and immediately became the vehicle for the publication of the most important physiological work in Bowditch's laboratory. In the years 1880 to 1883 two important articles were published: one by Bowditch and Southard, and another by Bowditch and G. Stanley Hall. Bowditch seems to have been the only physiologist that had observations published from his laboratory in the years that I have mentioned. The above is not a fair summary of the work done there during this period, for the research on the vasomotor nerves by Bowditch and Warren was commenced, I believe, in the old building on North Grove Street, and it is possible that Ernst was beginning his bacteriological work there in 1883, before the new medical building was completed, but it certainly had not advanced far. The three or four years after Bowditch moved into his new laboratory spanned the golden period of his scientific research in America, and it was my good fortune to see much of that work, and to be intimately associated with him in his laboratory. After 1890 I am unaware of any major scientific work which he published under his own name. Probably his main ambition had been to give physiology standing and resources in this country, and when he saw these grand objects largely achieved he was content to lay down the oars, and let the boat glide along under its own momentum. He had been a good physiologist, and did important work that may well serve as a good example to those that follow him. He was a good and faithful teacher, but I think that that kind of occupation irked him after a while, but he had well earned the satisfaction which he must have felt when he saw the magnificent group of buildings arise on Longwood Avenue, due largely to him, and when he knew that Boston had become one of the great medical centers of the world.

In the year 1883 Harvard Medical School celebrated its centennial anniversary, and dedicated its new building on Boylston Street. I went back to attend the celebration three years after leaving school. I was especially anxious to see the new physiological laboratory, and when I saw it I was captivated by it, and worked in it for three years thereafter. Bowditch had designed it with loving care, and had utilized every spot and crevice in it. It was almost regal in comparison with the Spartan quarters in the grim building on North Grove Street from which he had just emigrated. No child was ever more delighted with his new toys on Christmas morning than Bowditch was with his new laboratory. A few weeks after I first entered it, as I was talking with Wallace, the mechanic, in the dusk of the late afternoon, we could hear Bowditch wandering from room to room in the gathering gloom, until he came where we were located and said that it was hard for him to leave the place because it was so attractive. He had great reason for his satisfaction, for he now had the best physiological laboratory in the country, and had just been appointed dean of the medical school that was generally thought to be the best in America. The building was not only attractive in itself, but was conveniently located in the newer part of Boston, in full view of Copley Square, as no buildings intervened at that time, and was surrounded by the Victorian architecture of the Back Bay, which was then unsmudged by bituminous coal. The building is sometimes called the "old" one by those who were sheltered by it at a later date than myself; but to me it still has in recollection all the youthful charms that it exhibited as a parvenu in the Back Bay.

When the new building was dedicated Oliver Wendell Holmes made the principal address, and in his final burst of eloquence he expressed the hope that as time rolled on it would stand where it was, foursquare and upright, as a monument to Aesculapius, and for the use of future generations of his disciples. I may not have reproduced the exact words of the eloquent doctor, but I have, no doubt, expressed his wish and expectation. The building is still there, but twenty-three years later the school picked up its effects and moved into the magnificent group of five other temples of Aesculapius, where it is now located. That simple statement tells most emphatically with what marvelous strides medical education, with physiology in the van, was advancing in the United States in that short period. The step from North Grove Street was a very long and important one. The year 1883 marked the beginning of a new epoch in the Harvard Medical School, and that same year, and a few more in the same decade, formed the culminating point in Bowditch's scientific career. He was then in the prime of life, and had become an acknowledged leader in his science. His new laboratory was probably the most important scientific center in Boston, and was the focal point for many visitors from Europe as well as the United States. He was filled with enthusiasm for research, and appreciation of the great opportunities that had come to him and his institution. Some of the best scientific work of his career was done during the first few years that he passed on Boylston Street, and then he merged himself in the work of his pupils and of the school as a whole.

Bowditch's administrative duties as dean of

the medical school must have absorbed a good deal of his time and energy during the first few years of the existence of the new building, which had then many distinguished visitors who wished to see both him and the attractive edifice which was his pride and joy. William Osler attended the dedication of the new building, and I think that it was a day or two later that I met him in the physiological laboratory, and was introduced to him by Bowditch. I had never seen him before, although we had had a slight correspondence on a subject of mutual interest. At that time he was a little known young professor of a miscellaneous aggregation of subjects known as the Institutes of Medicine, in McGill University, Montreal. The institutes in his case meant histology and pathology, with physiology thrown in gratis. Although he lectured on physiology his practical work was largely microscopic, for it was recorded that he was somewhat diffident in the presence of a kymograph. During his visit to the laboratory a friend remarked to him that it was impossible for a laboratory worker to engage in practice. There was a large element of truth in this statement, as anyone who has tried the combination of practice and research will be willing to admit, although some resolute and indefatigable individuals like John Hunter have succeeded with both, even though Hunter sometimes vented his displeasure in a pungent remark when he was obliged to go forth to earn his guinea.

But it is frequently possible for a man to exchange his laboratory for the general public, and become a conspicuous success in practice, and we have no more shining example of this kind of man than Osler. For ten years he broadened the foundations of his future work by the study and teaching of physiology and pathology in a medical school, and during this time the public passed him by, and very little private practice came his way. For a part of these ten years he was a visiting physician to a hospital, and a hospital ward may be a kind of laboratory. In less than a year after I met Osler in the Harvard laboratory he was called to a professorship of medicine in Philadelphia. Five years later Johns Hopkins had him, and not long after that the whole world knew him, and physiology and pathology had their triumph.

For a little time after I became an inmate of Bowditch's laboratory he was fully occupied with his new duties as dean, and as I had no very definite ideas regarding a problem to work on, I was obliged to wait for suggestions from him. I began some work in the pathological laboratory of William F. Whitney, and continued it for a while, but dropped it after I became fully occupied with physiological activities. Bowditch's assistant at this time was Joseph W. Warren, who became associated with him in 1881. Warren was born in Springfield, Massachusetts, and received his medical degree in Germany. I do not think that he ever engaged in practice. He gave some instruction in elementary laboratory work to the undergraduates, and ably seconded Bowditch in his researches, but did very little independent investigation. Bowditch and Warren had evidently begun their well-known work on the vasomotor physiology of the cat in the old medical school, and they brought their apparatus to the new one, where they were soon ready to continue their plethysmographic research.

At the suggestion of Bowditch I undertook an investigation of the vasomotor physiology of the frog. I began with a microscopical study of the circulation in the web of the curarized animal when the peripheral part of the cut sciatic nerve was stimulated in various ways. A small artery of the web was observed under the microscope, and the diameter of the vessel measured with a micrometer eyepiece while the nerve was being stimulated. The observations were made at regular intervals, and their duration measured with the metronome, and then a graph of the results was made. Satisfactory results were obtained in these experiments, but the work was very fatiguing, and I soon resorted to the plethysmograph, with which permanent and continuous records were easily made. The frog soon proved to be a most convenient and satisfactory subject for this kind of experimentation, as it could be kept for days under curare, and required no artificial respiration. As the piston-recorder for air transmission had not been invented then, I used very small and delicate Marey drums for recording the changes in volume of the hind leg. The results of my vasomotor experiments were very similar to those obtained by Bowditch and Warren in their work with the cat. Contraction of the blood vessels was easily excited with tetanic stimulation, and dilatation was frequently obtained with slowly repeated single-induction shocks of appropriate strength, but dilatation after stimulation as the effect of degeneration of the nerve was doubtful. I found on microscopical examination that the frog's sciatic nerve degenerated very slowly, if at all, after cutting. I also made a very careful study of the cut nerves of the cats used in Bowditch and Warren's experiments, and found advanced degeneration after the third day, and my report of my findings was published as an appendix to their paper.

Although Bowditch and his assistant discovered nothing entirely new in their research, they systematized and corroborated what had already been

discovered regarding the vasomotor nerves of the limbs, and introduced an accurate method of studying and recording their activity. The apparatus for recording was practically the same as that employed by Mosso in studying the pulse, and described and illustrated by him in his monograph, *Die Diagnostik des Pulses,* published in 1879, but was modified to control the temperature of the water in it. The great value of the research was largely due to the deliberation and meticulous care which Bowditch and his assistant devoted to it, for they did not publish their results until four years later. That research became a classic, and the only criticism to which it is open, and one that applies to my own as well, is that the secondary coil of the induction apparatus should have been calibrated with a ballistic galvanometer in order that more exact data regarding intensities could have been given. The ubiquitous dry cell had not come into use in those early days, and whenever I needed an electric current I was obliged to go to the battery closet in the gallery of the laboratory and set up the Grove cell which supplied the central station on the floor below. Bowditch was proud of that little wooden tower, which housed the variable interrupter, and where all the electric wires of the laboratory converged, but its use did not contribute to easy working. There were no electric street currents for the distribution of light, heat and power in those days, and consequently there were no electric lights, and none of the convenient little electric motors that are now so useful in laboratories of every description. Bowditch's artificial respiration apparatus was run by a water motor with water supplied by a tank in the upper story of the building, but he loved that apparatus and showed it with pride to the numerous visitors whom he conducted about his premises. He had a regular itinerary, which included a visit to my frog, — when I had one working, as I usually had, — and I am inclined to think that most of the visitors were more thrilled by the sight of that insignificant animal recording its secrets on the smoked drum than by anything else that they beheld in the establishment. I recall a French count who spoke no English, and about whom it was whispered that he was a great duelist, who was especially moved at the spectacle.

As Bowditch was dean at the time about which I am writing, it fell to him to receive the visitors who wished to see the new building. He did double duty then, both as dean and as a physiologist who was decidedly proud of his laboratory. I think that there were only few of such visitors who were not shown the laboratory. I remember seeing there Sir Robert Ball, the Astronomer Royal of England, who was no doubt attracted to the place from his desire to meet the grandson of the distinguished Nathaniel Bowditch, the mathematician and navigator. One day Lord Rayleigh, the eminent English physicist, and Lady Rayleigh were conducted about the laboratory by Bowditch. John C. Dalton, the physiologist, appeared there on one occasion, and Bowditch brought him to my table and introduced me to him. I had studied his textbook before I entered the medical school, and I regarded him as a sort of physiological patriarch, although he was then twenty-two years younger than I am at the present moment. Any awe that I may have felt was soon dispelled by astonishment when I saw him take a cigar from his pocket, and with the permission of Bowditch light it, and saw for the first time in history that laboratory desecrated with tobacco smoke; for smoking was strictly forbidden there.

Bowditch's personal friends were frequently seen in his laboratory, and some of them called him Henry. William James and Stanley Hall sometimes appeared, and professors in other departments had business there. Two visitors who came several times, and who made a particular impression on me, were John S. Billings and S. Weir Mitchell. There seemed to me to be an especial bond that drew Bowditch and these two men together, and I think it possible that the bond was their war experience, but I never heard Bowditch speak directly of that experience. Once in a lecture to my class in my undergraduate days he stated that he had often slept while he was traveling on horseback. This statement no doubt referred to the time when he was a cavalry officer in the Civil War, but he did not say so, and I never heard him allude directly to the Civil War. I remember that a military procession passed through Boylston Street on one occasion, which was probably Memorial Day. As Warren and I watched the soldiers through the laboratory window, Bowditch stood near by and looked at the company without any comment. He could be reticent, and was always modest. Not long ago I consulted the full and careful notes of his lectures which I made in 1877 and 1878 to find what he said about the all-or-none properties of cardiac muscle at this time. What he said was brief. He stated that the extent of the contraction of voluntary muscle corresponds within narrow limits to the intensity of the stimulus, but that contraction of the muscle of the heart under constant conditions does not vary in this way. He did not mention that it was he who made the interesting discovery regarding the heart.

Weir Mitchell was interested in the work of the laboratory, and was searching for information

when he came to it, for he was always at heart a physiologist. He was kind and gracious to the youngest inmate of the laboratory, and when I published my contribution from it he sent me a message of approval which I greatly appreciated. The article was published in 1887, but the work was begun in 1883. In those days we did not hasten publication in order to get a job, as the jobs did not exist. However, I did other work during this time, the greater part of which was in bypaths from the road I had been traveling. While I was working in Bowditch's laboratory I was in the country at frequent intervals, as well as during the summer vacations. I had a laboratory of my own in which I worked when living at home, and which I regarded as an extension of the Harvard laboratory, and most of the work that I did there was credited to Bowditch's laboratory, as my problems were intimately connected with his at that time. The piston-recorder for air transmission was invented and first used in my own laboratory before I applied it to my work in Boston; it was there that I first used photography for making graphic records of physiological phenomena, and I am inclined to think that I was among the earliest to employ this method in this country.

At the same time that I entered the Harvard laboratory Harold C. Ernst, who was a year ahead of me in the medical school, set up his incubator for cultivating tubercle bacilli in the chemical hood in the gallery of Bowditch's laboratory. This was the commencement of his career in bacteriology, and it was very nearly, if not quite, the beginning of the study of pathologic bacteriology in this country. Bowditch allowed him to continue his work for a considerable time, and then informed me that he was getting uneasy about the proximity of Ernst's bacilli, and quarters were found for him in another part of the building.

In the spring of 1884 Bowditch entertained the members of the Massachusetts Medical Society in his laboratory at the time of the annual meeting in Boston. He was to receive the members in the middle of an afternoon and exhibit a number of physiological experiments in full blast. It was a sort of débutante party for the laboratory. Bowditch summoned all his friends in the neighborhood capable of conducting a physiological experiment and had them on hand to entertain and instruct the members of the venerable society. Warren took charge of the cat experiment, my frog was working as usual, Wadsworth, the ophthalmologist, had a pretty experiment in physiological optics, but I do not remember the other demonstrations save one, which was memorable. Bowditch had pressed his old assistant, Garland, into his service, and they were to do a blood-pressure experiment on a dog. Just before the time of opening the exhibition they set to work to get their experiment under way. As my frog could be left to its own devices for a while, I paused before their table to watch them connect the manometer, and as I did so I noticed that someone had left a stopcock open, and that about all the blood in the animal had run out. Bowditch made a vigorous remark which I shall not record, and left the room in a rage, leaving his assistant and me to mourn the demise of the unfortunate dog. To all appearance he was as dead a dog as was ever seen in that or any other laboratory. The blood pressure had fallen to zero; he did not breathe; there was no pulsation of the mercury in the manometer. As some sort of action seemed called for I suggested that we give the inanimate body a hypodermic of ammonia, which was done. I do not know just how strong that ammonia was, for it was taken from a reagent bottle, but it was sufficient. After some patient waiting the animal gave a little sigh, and later began to breathe more vigorously; the heart commenced to beat, and the mercury to go up, and after a while we had a live dog again, although the blood pressure attained only about two thirds of its original height. Bowditch was summoned from his library, and took his place at the table without any comment on the transformation that had taken place, although I had witnessed one of the most interesting and instructive experiments of my experience. I have had some difficulty explaining to myself just what the ammonia did to that dog while it was devoid of respiration and circulation, but I have always retained a great respect for ammonia as a cardiac stimulant.

At the end of the academic year of 1884 Bowditch left his laboratory for a year's vacation in Europe. Warren delivered the lectures on physiology during the next year, but with what success I do not know, as I did not attend any of them. As he had charge of Bowditch's notes he no doubt made good use of them. I continued my work in the laboratory, and, as formerly, made it my headquarters when in Boston. I think it was at this time that Warren engaged in his independent research on the action of alcohol, in which I sometimes rendered a little manual assistance, but I hasten to say that Warren always drank the alcohol.

In the spring of 1886, after I had completed my work on the vasomotor nerves, Bowditch asked me to engage in experiments on the action of ether on the sciatic nerve of the frog. I was oc-

cupied with this research for a considerable time, and the results that I obtained are hard to reconcile with the present all-or-nothing theories of nerve conduction and muscle activity.

Bowditch was not a general biologist like H. N. Martin, who commenced to teach biology and physiology in Johns Hopkins University in 1877. Martin's early experience at Johns Hopkins duplicated closely that of Foster at Cambridge, England, for both were responsible for the teaching of a number of subjects which are related or basic to physiology, as well as physiology itself, and both founded schools covering the whole field of animal biology. This Cambridge characteristic of Martin's department at Johns Hopkins was in decided contrast to that of Bowditch's department in the Harvard Medical School. Bowditch was only concerned with physiology, and he was even something of a specialist in that science. His work and interest was in that division of physiology which was in the closest contact with that of the master in the Leipzig laboratory. He is said to have studied histology with two of the best French and German teachers of that science, but although he made some necessary allusions to microscopic structure in his lectures, I cannot recall, in my three years of intimate association with him in his laboratory, ever seeing him making any independent use of the microscope. He was obliged to advert to some chemical matters in instructing his class, but he unblushingly disclaimed any interest in chemistry, and attributed that indifference to the fact that he was color blind, and could not distinguish the colors of precipitates. This excuse always impressed me as decidedly thin, for John Dalton, the founder of modern chemistry, was color blind and gave his name to that defect. In physiology Bowditch stuck closely to his last, and never attempted to do anything but peg his kind of shoe, and having finished the shoe to his satisfaction, he was even willing to turn the hammer over to others and let them peg the other shoe. But the work which he did perform was that of a thorough and well-trained workman. The number of researches in which Bowditch personally engaged were not very numerous, but they were conducted in a very conscientious manner, and might well serve as patterns to some of the younger generation who are sending to market too much immature veal, instead of the heavy beef for which the scientific public is yearning. There is too much bread-and-butter research, at the present time, on the part of those working for a position, and too little work that is actuated by an irrepressible urge to question Nature.

After 1886 I did no more work in the Harvard laboratory, although I sometimes saw Bowditch there or elsewhere, and sometimes heard from him by letter. I commenced practice in 1887, and became so thoroughly immersed in ophthalmology that I had little time for anything else. My physiological work became more and more restricted to the physiology of vision and physiologic optics and closely related subjects.

In the summer of 1887 I received a letter from Bowditch stating that a movement had been started to establish a physiological society, and inviting me to join in it. I remember that it was hot weather then, for I asked that the place for the meeting be one where cooling drinks could be obtained, but I heard nothing further of the matter until the first meeting of the Society was called in New York, in December of the same year, for organization and the election of officers. I attended that meeting, of which Weir Mitchell was made chairman and Martin secretary. Seventeen men were present, and all were either actively engaged in physiological work or in closely related sciences. This was the beginning of the American Physiological Society, whose semicentennial anniversary was celebrated in Baltimore last April. These seventeen men represented the twenty-eight original members, eleven of whom were not present at the first meeting. Of the original members only five survive; all of them attended the organization meeting in 1887, and four were present at the recent celebration in Baltimore. The United States and Canada were pretty thoroughly combed to make the original group of twenty-eight members, and only about half this group were professional physiologists. The other original members were not actively engaged in physiological research, but regarded it as very essential for the advancement of biology and medicine. The society founded then has become one of the great scientific associations of the world, and the parent of the American Society of Biological Chemists, the Society for Pharmacology and Experimental Therapeutics and the Society for Experimental Pathology. The combined membership of the parent society and of the organizations which parted from it, but are still closely affiliated with it, numbers more than twelve hundred. The leap from twenty-eight to twelve hundred demonstrates most vividly what has been accomplished in physiology and scientific medicine in America in the last fifty years. Bowditch was one of the most active founders of the society, and lived long enough to enjoy the satisfaction of knowing that he had contributed much to a second great factor in the development of biology and medicine in this country.

I continued to visit Bowditch's laboratory for a while after the foundation of the American Physiological Society, and sometimes met him at its meetings, but our ways diverged more and more until I lost my health and gave up practice. As I was recovering from my protracted and very severe illness I learned that Bowditch had resigned his professorship, and I knew that he too was ill, and as a scientist had faded from the picture. A flood of memories of the happy times I had spent in his laboratory impelled me to write to him. I was not able to write myself, so I dictated a letter as I lay on a couch. I told him how much the three years that I spent in his laboratory had meant to me, and how much I regretted that he could no longer pull the stroke oar in his institution. He sent back a very kind, but a pathetic letter. He seemed to be philosophically resigned to his affliction. He typed the letter, he said, because his shaking hand would not allow him to write. He seemed greatly pleased that the authorities of the medical school had given him a room in the new building which he had helped to erect, and he hoped to haunt the institution for a while longer. He expressed a desire that we should meet again in Boston, but we never did. When he departed, a fine gentleman of the best New England type left the Harvard Medical School greatly his debtor, and also left medical education in this country on the high plane that he used to dream about under the rafters of the old medical school on North Grove Street.

Henry Pickering Bowditch was extremely fortunate to be born in the time and place to which fortune assigned him. When he began his work in the Harvard Medical School the resistless tide of modern science was about to overtake medicine, and to urge it forward with ever-increasing speed. The virgin resources of our country were being rapidly converted into wealth which would soon be at the disposal of the Harvard Medical School, where it could be used intelligently and effectively for the good of humanity. The hope of the world now lies largely in scientific medicine, and the Harvard Medical School is still devoting its high ideals and great resources to its advancement.

PRIMARY CANCER OF THE LUNG*

DONALD S. KING, M.D.†

BROOKLINE

ABOUT 400 B.C., Hippocrates advised patients suffering from consumption to "buy a cow, drive the cow to the mountains, and live off the cow." Hippocrates knew his pulmonary tuberculosis, but did not recognize pulmonary cancer.

By 1815 A.D. Laennec, by means of stethoscopic examinations checked by postmortem observation, had learned much more about tuberculosis and pneumonia than Hippocrates could possibly have known, but cancer of the lung was not within his clinical ken.

The year 1842 is the date of the first record at the Massachusetts General Hospital which gives the history, physical signs and postmortem findings in a case of "scirrhous malignancy" of the lung. The complete and classical picture is given in this early record — a patient of forty-four who lived for six months with very severe and persisting right-chest pain referred to the shoulder. There was "great distress for breathing," severe paroxysmal cough and, at the end, hemoptysis.

Apparently in those days the physician who followed the case also did the postmortem examination; in one case, the autopsy note is preceded by the following apology: "This man entered with signs of phthisis, but he was so foolish and dirty that I made few examinations."

In the case recorded in 1842 there was no need for such an apology, as frequent physical examinations were made and there were found to be constant unilateral wheezing and "dullness on percussion over the back where the vesicular respiration was scarcely heard." Let me remind you that these are the cardinal signs of bronchial obstruction, although not universally recognized as such by physicians until almost a century later.

In the early records there are some apologies, too, for the type of postmortem examination performed, such as: "Examination was somewhat hurried expecting opposition." In the 1842 cancer case, however, the autopsy also was careful and complete. The malignant growth was found to involve the membranes of the trachea and bronchi. It surrounded and narrowed the bronchial tree, infiltrated the pulmonary tissues of both lungs and metastasized extensively to the neighboring lymph nodes.

*Presented at the annual meeting of the New Hampshire Medical Society, Manchester, May 17, 1938.

†Associate in medicine, Harvard Medical School; associate physician, Massachusetts General Hospital.

In 1856, in the *Boston Medical and Surgical Journal,* C. D. Ellis reported a similar case. It is interesting that he said: "Physical examination, to a certain extent, indicated tuberculosis, but findings in the lower part of the back [which he had previously described as 'respiration — very obscure'] would not admit of this diagnosis." He added: "Long persistence of other thoracic symptoms and their character indicated grave disease of a malignant nature." He ended his autopsy report with this interesting statement: "If there be cells peculiar to a cancerous disease, they were found in this case."

Unfortunately, such excellent examples of clinical acumen were rare, and from 1875 to 1914 only 45 cases of pulmonary cancer appear in the hospital records. It was not until modern methods had made the diagnosis comparatively easy that large series of cases began to be reported and that thoracic surgeons took action. Even as late as 1920 a visiting physician at the Massachusetts General Hospital made the following case note: "The pathological report is definitely adenocarcinoma, which may be primary in the lung. This is so rare, however, that barium studies must be made" — in order to rule out primary cancer elsewhere in the body.

These modern diagnostic developments have all come in the present century: first the chest x-ray; then the bronchoscope; then chest surgery; and finally, in the decade 1920-1930, the combined efforts of the men working in these fields and of internists to establish the criteria for recognition of bronchial obstruction of varying degrees, accompanied by the realization that the one great cause of chronic bronchial obstruction is cancer.

The effect of these studies on the number of cases diagnosed at the Massachusetts General Hospital is best shown on a chart (Fig. 1). As will be noted, a sharp increase occurred after 1914, a sharper one after 1920, and in the year 1936 we studied 62 cases, in 1937, 51. We now have records on 475 cases in which the diagnosis was made clinically, and in 158 of these it was proved by histological examination — in 72 by autopsy, and in the others by bronchoscopic biopsy, biopsy of metastatic nodes or exploratory operation.

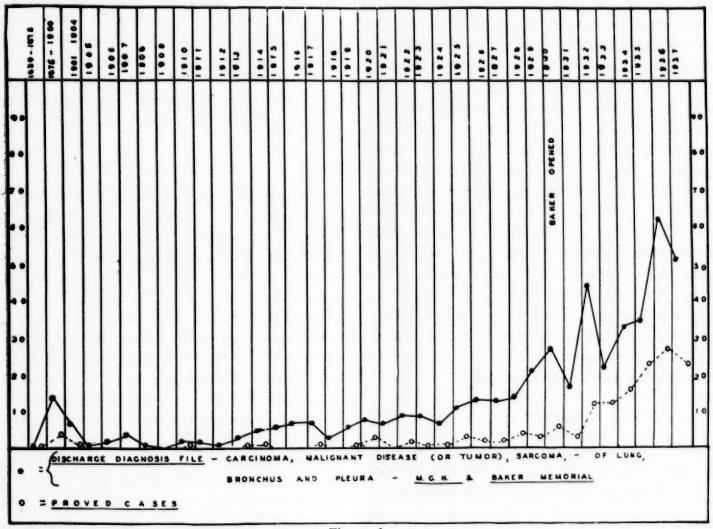

Figure 1.

Does such a chart indicate a sudden increase of pulmonary cancer in the population at large? Many authorities maintain that it does, citing as evidence the fact not only that autopsy reports show an increase in the number of cases of pulmonary cancer coming to autopsy, but that the percentage of pulmonary cancer to malignant tumors in other organs has risen rapidly, so that in some hospital reports 10 per cent of all cancer is primary in the lung.

Against this claim must be balanced the obvious fact that changing hospital autopsy per-

centages may depend on the changing character of the hospital population. Largely owing to the x-ray, interest in lung conditions has increased tremendously, and now everyone who coughs twice demands a chest film. By such studies many cases of cancer are discovered and now sent to a hospital, where thoracic surgery offers some hope of cure.

I take it, then, that you will agree that at least at the Massachusetts General Hospital pulmonary cancer is common. If this be so, you may meet it in your practice, and I assume that you are interested in a little fuller discussion of the good old triad of diagnosis, prognosis and treatment.

As I have already hinted somewhat broadly, the most important point in diagnosis, and in the understanding of the clinical course of the disease, is recognition that so-called pulmonary cancer is bronchiogenic in origin; in 70 per cent of all our cases the origin was in the large bronchi and not in the small branches. This point I wish to develop a little further, and shall go on to show that although pulmonary cancer originates in the bronchi and gives varying degrees of bronchial obstruction, it is not content to stay there, but characteristically invades the neighborhood or wanders to distant parts of the body. In 9 per cent of our cases the first symptoms were from such metastases, and not from the primary growth itself. In passing, I might state that 80 per cent of all reported cases occurred in men, usually but not always of the cancer age. There were rare cases in the twenties, but none below twenty.

Diagnosis

We shall first take up matters of diagnosis, noting especially how in each procedure we depend on evidence of obstruction of the bronchi.

Symptoms

Unfortunately there are no symptoms pathognomonic of pulmonary cancer. The list is practically the same as for any chronic lung condition. In our series, as in most others, the symptoms arranged in the order of frequency are: cough, pain, weight loss, hemoptysis, dyspnea, fever and purulent sputum.

Persistent pain, especially if not of the typical pleural type, is a very important symptom, often sufficient to suggest the diagnosis. Dyspnea is a much commoner symptom of cancer than of tuberculosis and is due, at least in part, to bronchial obstruction. The frequency of hemorrhage is also due to the predominant involvement of the bronchial mucous membranes, with resulting ulceration and bleeding into the bronchial tree.

Physical Examination

As noted in the early Massachusetts General Hospital cases, the distinctive physical signs of this condition are wheezing and diminished or absent breath sounds. In our series, wheezing was found in more than 33 per cent of the cases, and was unilateral in one third of these. It is a sign of partial bronchial obstruction. If the obstruction is complete, atelectasis, or collapse of the lung supplied by that bronchus, results and the breath sounds are absent from the area of flatness.

We are learning that in pulmonary tuberculosis involvement of the bronchial membrane by tuberculous infiltration is not uncommon, and that partial or even complete bronchial obstruction may result. Nevertheless, the characteristic signs of tuberculosis are those of consolidation and cavitation, not those of bronchial obstruction. So in cancer the physical signs which are most helpful are those of varying degrees of bronchial obstruction.

X-Ray

Much as he hates to admit it, the internist is dependent on the roentgenologist in almost every case of lung disease. The x-ray shadows of pulmonary cancer can be classified in many ways, but in general they are of two types; hilar and peripheral.

In the hilar type a shadow is cast by the tumor at the lung root itself, or by the atelectatic lung resulting from the bronchial obstruction due to such growth in the larger bronchi. In our series, as noted above, 70 per cent of the cases were of this type. Peripheral tumors show as rounded or lobulated shadows situated away from the lung root, but not necessarily far out toward the periphery. These numbered 10 per cent of our series. Present-day lateral x-ray films are particularly important in localizing these tumor masses. Until a few years ago the discrete tumors were usually regarded as metastatic, but recent studies have shown that in many cases they are primary bronchiogenic cancers, starting in the smaller bronchi rather than in the main bronchial tree.

Besides the hilar and peripheral tumors, 10 per cent of our cases when first x-rayed showed a pleural effusion sufficient in amount to obscure the underlying pulmonary disease. In the cancer age particularly, malignant tumor must be ruled out in all cases with pleural fluid. In 10 per cent of our cases no x-rays were available.

Bronchoscopy

From the bronchoscopic standpoint, it is particularly fortunate that pulmonary cancer originates so frequently in the large bronchi. Our

series started before the day when bronchoscopy was a common procedure, and the percentage of cases so examined is not so large as it would be in a series started today. Only half our cases were bronchoscoped, and slightly more than one-half of those so studied showed a tumor from which a specimen could be taken. However, Chevalier Jackson and other bronchoscopic specialists report that 60 or 70 per cent of all cases of pulmonary cancer can be diagnosed by means of the bronchoscope, and I am inclined to believe that this is not an exaggerated statement.

Other Procedures

Symptoms, physical signs, x-ray shadows and bronchoscopy are, then, dependent to a large extent on the fact that cancer of the lung is bronchiogenic in origin. We shall, however, note a few other diagnostic procedures not dependent on bronchial involvement.

In 15 per cent of our cases a correct diagnosis was made by biopsy of cervical or axillary lymph nodes. A very careful examination of the neck and axillas should be made in all suspected cases.

Occasionally a large tumor is situated near enough to the chest wall to justify tapping with a large needle and obtaining a so-called "suction biopsy." This procedure should be applied with caution, but in selected cases is preferable to bronchoscopy and may establish the diagnosis.

We find that in the modern hospital sputum examination has fallen somewhat into disrepute, but it is obvious that in all chronic lung conditions the most important examination, perhaps next to the x-ray, is that of the sputum for the presence or absence of tubercle bacilli. It must be realized that there are reported many cases in which tuberculosis and cancer are associated, and that the presence of either does not exclude the possibility of the other.

Differential Diagnosis

Space forbids me to discuss differential diagnosis in detail. I have mentioned the confusion of pulmonary cancer with tuberculosis, and call particular attention to the danger of confusing bronchiogenic carcinoma and the so-called "benign adenoma" of the bronchus. There is still some question as to whether these adenomas are really histologically benign, and it is still true that a histologically benign tumor may cause death by bronchial obstruction with suppuration; but before one gives the essentially hopeless prognosis which attends a diagnosis of cancer of the lung one must be sure that the pathologist is not being confused by a carcinoid type of tumor which has been found to be extraordinarily slow in its rate of growth.

Complications

The complications of pulmonary cancer are also dependent on its bronchiogenic origin. Any bronchial obstruction is apt to be followed by suppuration in the bronchi or pulmonary tissues beyond this point. Bronchiectasis and pulmonary abscess occurred in 12 per cent of our series. However, purulent sputum was present in 33 per cent, and some degree of bronchial infection was undoubtedly present in a great majority. Necrosis of the tumor itself is a not uncommon finding.

Although the primary tumor originates in the bronchus, the growth characteristically spreads outside this area. The tumor may infiltrate neighborhood structures, particularly the lung tissue itself, and either by extension or metastasis to lymph nodes may cause pressure on nerves — the recurrent, phrenic or sympathetic nerves — or may involve the esophagus, diaphragm or pericardium.

In 14 per cent of our cases there was proved nerve involvement. As a rule the cancer metastasizes early. There are numerous statistics as to the site of metastases.

The order of frequency in our 72 autopsied cases was as follows: bronchial lymph nodes, other lymph nodes, adrenal glands, liver, bone, brain, kidneys and pericardium. There were metastases to the lymph nodes in one half the cases, to the adrenals and liver in one third, to the bones and brain in one fifth and to the kidneys in one sixth.

Prognosis

As in all cases of cancer, the outlook is poor. We have not yet had a chance to make a careful follow-up of all our 158 proved cases. One hundred and five of these patients are dead, and the average duration of life from the first reported symptoms was 9.3 months. This agrees with reports from other clinics, although the average duration is spoken of by different writers as being from six months to two years. Of our 105 fatal cases, 19 showed symptoms for over one year and only 4 for over two years. At the last report, of the remaining 53 patients who are either living or not known to be dead, 5 had had symptoms for over one year and only 6 for over two years.

It is worth while to mention here the different cellular types of pulmonary cancer and the relative duration of life in each. In our series the average duration of life for the epidermoid or squamous-cell carcinoma was 9.6 months, for the adenocarcinoma 7.5 months, and for the oat-cell or undifferentiated type of carcinoma, previously often confused with sarcoma, 5.6 months. There is a fourth group reported as carcinoma but not otherwise classified, with an average duration of 12.6 months.

These figures show that the oat-cell or undifferentiated type of tumor is more rapidly fatal than the other types, but that the duration of life in any event is short.

Treatment

X-Ray

There is some difference of opinion as to the effectiveness of roentgen therapy in pulmonary cancer. I can only give you the experience at the Massachusetts General Hospital, and the opinion of our roentgenologist, Dr. George W. Holmes.

Forty-three of our 158 proved cases have had x-ray treatment. Many other cases which undoubtedly were those of cancer but which could not be histologically proved to be so have also been treated in this way. In no case has anything been accomplished that could be considered a cure. However, the average duration of life in the x-ray-treated patients who died was 15.4 months, as opposed to the average of 9.3 months for the whole series. You may draw your own conclusions.

Dr. Holmes has found that the oat-cell or undifferentiated type of carcinoma responds more favorably to x-ray than either the squamous-cell type or adenocarcinoma. There is some danger that x-ray treatment may hasten a beginning suppurative process. Be that as it may, indiscriminate x-ray treatment of lung tumors should be discouraged, and whenever possible a histological diagnosis should be obtained before treatment is started.

Surgery

In the past ten years, and particularly in the past six, thoracic surgeons have offered the hope of cure of cancer of the lung by its surgical removal. The procedure involves the removal of one or two lobes or an entire lung. After decades of hope, this procedure has now been made technically possible, and it has been shown that patients can live in reasonable comfort with an entire lung removed.

Nevertheless, pitfalls beset the surgical program. In the first place, metastases to the regional lymph nodes or far beyond them may have taken place by the time the patient is first seen, and except in cases where the removal of the regional nodes eliminates all probable spread of the disease, operation will of course prove fruitless. In some of our cases the lung has been successfully removed, only to have the patient die some months later of brain metastases which were silent at the time of the operation.

In January, 1938, Dr. Edward D. Churchill, of the Massachusetts General Hospital, reported his operations up to that time. Eighteen cases were selected for surgical resection. In 11 of these an entire lung was removed; in the other 7 one or more lobes were removed. Of the 18 patients, 5 are still living and 13 are dead — 7 deaths resulting from operation and 6 from extension of the disease in spite of it. Of the 5 patients now living, 1 has symptoms of brain metastases, 1 has a very troublesome empyema as a postoperative complication, and 3 are apparently well, having been operated on two or three years ago.

As an internist I still have faith in the surgical treatment of pulmonary cancer, although I realize its limitations. I see no excuse for surgery in cases where there is no chance of excising the entire growth, but I do believe that every effort should be made to establish an early diagnosis, and to have a particularly well-trained thoracic surgeon remove the growth where there is a chance of doing so, even with regional metastases.

Besides the 18 cases in which removal was attempted, Dr. Churchill explored 10 cases and found an inoperable condition in all. Such a procedure is of course justified with present-day thoracic surgery, as in abdominal surgery.

Summary

Primary pulmonary cancer is of frequent occurrence. Fifty-one cases were studied at the Massachusetts General Hospital in 1937 alone. The disease is bronchiogenic in origin and may belong to any one of three cellular types: the squamous-cell carcinoma, the adenocarcinoma, or the oat-cell or undifferentiated carcinoma.

Eighty per cent of the cases occur in men, usually, although not always, of the cancer age. The most distinctive symptoms are persistent, nonpleural pain and dyspnea. Common physical signs are wheezing, which may be unilateral, and diminished or absent breath sounds.

There are two main x-ray types — the hilar, with or without atelectasis, occurring in 76 per cent of our x-rayed cases, and the peripheral occurring in 12 per cent. Bronchoscopy will establish the diagnosis in 60 to 70 per cent of cases. Suppuration is a common complication. Pulmonary carcinoma invades the neighboring tissues and metastasizes to the regional lymph nodes. The recurrent, phrenic and sympathetic nerves may be involved. Distant metastases are frequent to other lymph nodes, adrenals, liver, bones, kidneys, brain and pericardium. The average duration of life is less than one year.

X-ray treatment offers no hope of cure and only slight hope of prolongation of life. In the

proper hands, surgery is worth trying in carefully selected cases.

1101 Beacon Street.

Discussion

Dr. M. Dawson Tyson, Hanover: Dr. King, in his customary scholarly and stimulating manner, has given us a complete survey of the subject of primary pulmonary cancer. There is nothing that I can add to such an excellent presentation. However, from our standpoint in New Hampshire perhaps I can bring the subject a little closer home.

There are two features of this disease that I shall discuss. One is the incidence of primary carcinoma of the lung, and the other its operability.

With respect to incidence, our experience duplicates on a small scale that of Dr. King at the Massachusetts General Hospital in that the number of cases diagnosed as cancer of the lung has increased tremendously since we have made a concentrated effort to detect this condition. Perhaps of more importance than our own efforts is the fact that the general practitioner is more alert than formerly in observing the signs of pulmonary cancer. As will be brought out, however, it often takes too long to arouse the doctor's suspicion that cancer of the lung is present. In our clinic, the final diagnosis of primary carcinoma of the lung was made only twice between 1930 and 1935. In one case, the diagnosis was substantiated by an autopsy; in the other, the diagnosis was made on the basis of the clinical and x-ray findings. Since July, 1935, when our thoracic service was organized, primary cancer of the lung has been diagnosed in 14 cases, and of these, 10 were referred in the last eleven months. In all but 2 of the 14 cases the diagnosis was proved by actual examination of the tissue obtained either at autopsy, at bronchoscopic biopsy, from aspirated pleural fluid or at operation.

From the standpoint of operability it is important to note that none of these cases were operable, though in 11 of the 14 cases symptoms pointing to pulmonary disease had been present from five months to a year before the diagnosis was made. The remaining 3 cases belong to that group in which either because of the rapidity of growth of the lesion or because it originates in a "silent area" the patient presents himself too late for surgery. In the 3 cases under discussion symptoms had been present for only one or two months. In 2 the first complaint was of a metastatic mass in the left supraclavicular fossa. In the other, cough had been present for one month, and hemoptysis for two weeks. The last patient died of pulmonary hemorrhage, and autopsy showed a very extensive, highly malignant pulmonary cancer. It is difficult to imagine how cases of this type can ever be amenable to surgical attack.

In the 11 cases mentioned above it seems reasonable to suppose that many if not all of them were operable at some time in the course of the disease when the diagnosis could have been made by modern methods. It is in this group where the lesion apparently progresses relatively slowly that every effort should be made for an early diagnosis if any results are to be expected from surgery. Every case of influenza or a cold followed by a chronic cough and fever should be investigated. The diagnosis of tuberculosis in the absence of a positive sputum should always be questioned, and a further study made. The diagnosis of "unresolved pneumonia" should be withheld until all possible attempts have been made to rule out other causes of the pathologic changes observed. The age of the patient is no criterion, as one of our cases was a woman of twenty-five with a six months' history who at exploratory thoracotomy was found to have a huge inoperable squamous-cell carcinoma of the left lung.

It is evident that primary cancer of the lung is a common disease, and this fact is becoming more and more widely appreciated by the medical profession. It is hoped that with this fact before us it will be possible to recognize the condition in its early stages so that the results of surgery can be accurately evaluated.

Dr. King: Of course, as we have presented the problem of cancer of the lung, it looks to you like a hopeless problem, and in general this is true. By the time the patient seeks medical advice, the average case of cancer of the lung is beyond hope, but we must still keep looking for the early cases, and certainly in a small percentage we shall be able to get cases in which a cure is possible. We must never be confused by average cases and average figures.

A Physician: Can Dr. King explain the high percentage of cancer of the lung in men and the very low percentage in women?

Dr. King: I cannot answer that. No one, of course, knows the cause of cancer. Everyone has tried to discover some irritant of the respiratory tract which might be responsible. The use of tobacco is always mentioned as a possibility, but it has never seemed to me to be a satisfactory explanation.

A Physician: Perhaps with cigarette-smoking on the increase among women the figures will change.

Dr. King: That is possible.

THE PROBLEM OF SYPHILIS ON THE WARDS OF A LARGE GENERAL HOSPITAL*

H. Houston Merritt, M.D.,† and Merrill Moore, M.D.§

BOSTON

THE purpose of this study was to attempt an analysis of the incidence and cost of syphilis to the in-patient department of a large general hospital. With this aim in mind the diagnostic files of the Boston City Hospital were studied for the diagnosis of syphilis during the ten-year period between 1926 and 1935.

At the outset it was realized that any results from the study would only be suggestive, since during this period there was no syphilis service at the hospital and there was no uniform method of diagnosing and cataloguing syphilis on the various medical, surgical and special services of the hospital. The number of cases here presented represents an absolute minimum because even though the cases are cross-indexed, each is listed separately and there is no duplication. On the other hand we are certain that many cases have been missed, since blood serological tests are not routinely performed on all services of the hospital.

Table 1 shows the total admissions to the hospital for the years 1926 to 1935 and the number of cases in which the diagnosis of syphilis was made. The admission rate for syphilis has practically doubled in this decade, but the percentage of syphilitic patients remains practically constant, owing to increasing total admissions.

Table 1. *Number of Patients Treated at the Boston City Hospital.*

YEAR	ALL PATIENTS	SYPHILIS PATIENTS	PER CENT OF TOTAL
1926	20,557	470	2.28
1927	22,819	584	2.56
1928	23,576	588	2.49
1929	24,939	740	2.97
1930	28,665	642	2.24
1931	32,020	654	2.04
1932	37,368	849	2.27
1933	39,979	834	2.08
1934	39,969	789	1.97
1935	39,012	835	2.13
Totals	308,904	6,985	
Average			2.26

An interesting sidelight on this study was that the average stay in the hospital for the patient with the diagnosis of syphilis was 20.7 days as compared with 12.6 days for the general hospital population. Thus the patients with the diagnosis of syphilis (regardless of whether or not their admission to the hospital was due to the syphilitic infection) cost the hospital approximately 64 per cent more than the average patient.

The cost of syphilis to the hospital, subdivided according to the organs involved, is shown in Table 2. In this table it can be readily seen that when the unclassified‡ group of patients is excluded, the manifestations of the disease which are of most serious economic importance to the in-patient department are certain late manifestations — those of the central nervous system and the cardiovascular system. The cost of handling these cases was over three fourths of the total cost. The next largest total cost was that due to primary and secondary syphilis; approximately 12 per cent of the cases were found in this group. Most of these cases were admitted to the hospital not so much on account of physical disability but as a public-health measure in order to give patients sufficient treatment to prevent transmission of the disease. The third most expensive category was that of congenital syphilitics. Approximately 5 per cent were in this group. The late manifestations of syphilis in the skin, bones, mucous membranes, eyes and internal organs were of relatively minor economic importance.

Table 2. *Cost of Syphilis to the Boston City Hospital for the Ten-Year Period 1926-1935 in Relation to Type of Disease.*

TYPE OF SYPHILIS	COST
Primary	$6,818
Secondary	32,946
Cardiovascular	90,248
Brain and spinal cord	163,689
Skin and mucous membranes	9,211
Bone	2,326
Eye	2,487
Respiratory organs (nose, throat and lungs)	1,377
Liver	2,653
Urinary organs	486
Congenital	16,107
Unclassified (positive Wassermann or Kahn tests)	382,853
Total	$711,201

Table 3 lists the cost by years for the handling of the patients shown in Table 2 and also the percentage in relation to the cost for the total hospital population.

From an analysis of the material in this study

*From the Neurological Unit, Boston City Hospital, and the Department of Diseases of the Nervous System, Harvard Medical School.

†Assistant professor of neurology, Harvard Medical School; assistant visiting neurologist, Boston City Hospital.

§Associate in psychiatry, Harvard Medical School; assistant visiting neurologist, Boston City Hospital.

‡This group is probably composed chiefly of patients with positive blood reactions or patients in whom the diagnosis of syphilis was made but was not the chief cause of their entry to the hospital.

it is apparent that though on the whole the patients with syphilis were handled very well in the hospital, many improvements can be made. The difficulties in handling these patients are many and various. Chief among them is the fact that there is no syphilis service. As a result there has been no uniform system for the diagnosis and treatment of patients suffering from syphilis and no proper social-service follow-up, which is absolutely necessary in these cases in order to be certain that the patient will continue his treatment in the outpatient department after his discharge from the hospital. Without proper follow-up the time and money spent in the diagnosis and treatment of the patient in the hospital is almost entirely wasted.

Table 3. *Cost of Syphilis to the Boston City Hospital for the Ten-Year Period 1926-1935 in Relation to Total Cost.*

YEAR	COST OF ALL PATIENTS	COST OF SYPHILIS PATIENTS	PER CENT
1926	$1,319,207	$42,281	3.2
1927	1,355,340	56,552	4.1
1928	1,547,286	63,041	4.0
1929	1,825,836	82,631	4.5
1930	2,149,427	65,571	3.0
1931	2,371,184	79,339	3.3
1932	2,229,312	86,308	3.9
1933	2,164,645	78,102	3.6
1934	2,218,431	74,613	3.4
1935	2,410,285	82,764	3.4
Totals	$19,590,953	$711,202	
Average			3.6

* * *

On the basis of these results we feel that certain recommendations can be made for the handling of patients with syphilis in a general hospital.

1. A special service or a division of the hospital staff is necessary to care for patients with this disease in a uniform and effective manner. This would naturally entail separate wards and a house staff. Since the economic importance of the disease lies chiefly in its destructive effects on the central nervous system and the cardiovascular system, the visiting staff should include internists trained in the study of cardiovascular and neurological diseases as well as a dermatologist, an ophthalmologist and a pediatrician.

2. Adequate social-service assistance is needed in obtaining the necessary contact with the source of the infection of the individual and in contacting other members of his family for examination and serological testing. In addition it is necessary to have social-service assistance to ensure the follow-up of the patient after discharge from the hospital in order that he may be returned regularly for treatment, without which the disease progresses.

The establishment of a syphilis service would ensure a uniform method of diagnosis and treatment of patients with syphilis. The diagnosis would be made only after consultation with the syphilis service, and if the patient were not transferred to the syphilis service, the treatment could be outlined by consultation. Examination and observation on the syphilis service would benefit the syphilologist in the outpatient department, since the results of such studies would make possible a more effective and economical treatment.

This study was completed with the assistance of the WPA project (No. 14546) for the study of syphilis at the Boston City Hospital, 1937-1938.

UNEXPECTED DEATH IN EARLY LIFE*

SIDNEY FARBER, M.D.†

BOSTON

IN EXPRESSING my appreciation for this invitation to speak before the Massachusetts Medico-Legal Society, I should like to include a word of gratitude to its members for the opportunity they have given me to add to my experience in this highly interesting field. I shall not attempt to cover the subject in an exhaustive manner; rather, I shall restrict the discussion to personal observations. These have been gathered at postmortem examinations performed both in and around Boston during the last ten years.

Unexpected death may be defined, for the purposes of this discussion, as that which occurs without warning in individuals supposed to have been in good health, or in patients suffering from diseases which ordinarily do not terminate in this manner. Since the investigation of deaths caused by violence or murder does not lie within the province of the general pathologist, I shall make no attempt to deal with the problems. It is clear that the same pathologic process may account for death whether or not the deceased was under medical supervision preceding the unexpected demise. In fact, if accidents are excluded, virtually all cases of unexpected death in early life fall within the category of "death from natural causes," if sufficient investigation is made. I shall therefore include in this discussion disease findings not ordinarily considered of medicolegal interest. In so doing, it is my purpose to place the problem of unexpected death in early life in its proper setting as a part of the pathology of early life.

A history, if obtainable, may be either valuable or misleading. It is well known that a history may be unreliable even when supplied by an intelligent person, and that it may be deliberately falsified in order to avoid blame. On several occasions I have found severe pneumonia of at least three to four days' duration in young children who were said to have been completely well until they were found dead in bed. In these cases we are faced with either falsification or negligence as the explanation for the discrepancy between the history and the autopsy findings. It is recognized that a complete autopsy is the only reliable method of discovering the cause of unexpected death. Such an examination includes gross and microscopic examination of all organs of the body, and all indicated bacteriological studies and chemical analyses. It should be mentioned that although a complete postmortem examination will reveal the cause of death in the majority of cases, there remains a group which cannot be cleared up by this type of examination, either because no significant morphologic changes can be found, or on account of our lack of knowledge concerning the action or existence of certain etiologic factors.

*Presented at a meeting of the Massachusetts Medico-Legal Society, February 2, 1938. From the departments of pathology of the Harvard Medical School and of the Infants' and Children's hospitals, Boston.

†Assistant professor of pathology, Harvard Medical School; pathologist to the Infants' and the Children's hospitals, Boston.

UNEXPECTED DEATH CAUSED BY A CONGENITAL ANOMALY

At times congenital anomalies of the heart and the great vessels bring about unexpected death. A boy (A-34-83) three weeks of age was brought to the hospital because of cyanotic spells associated with convulsions of one week's duration. On the night of entry the patient choked while feeding and became quite cyanotic, but he did not appear to be critically ill. The next morning he had another cyanotic attack and died. Postmortem examination revealed a congenitally malformed heart. There was a defect in the interventricular septum. The most important finding was an anomaly of the left pulmonary artery, which branched higher than is normal and passed between the esophagus and the trachea, to be tightly applied over the posterior surface of the trachea at the level of the bifurcation. The malposition of the artery created the gross effect of a rubber band compressing the trachea at that point. Bronchopneumonia of the aspiration type was found. In this case the congenital anomaly was the cause of the difficulty in feeding and the aspiration of food. The effect of the anomalous left pulmonary artery plus the aspiration of food brought about fatal respiratory obstruction and death, which could not be clinically explained on the basis of the general condition of the child.

Serious respiratory obstruction may be caused by a congenital anomaly of the aorta and its main branches when, because of the persistence of a right aortic arch, a vascular circle is formed around the trachea and esophagus. Massive cerebral hemorrhage may cause death in patients known to have congenital heart disease. Subacute bacterial endocarditis, a complication which is a constant threat to children suffering from congenital heart disease, may be the source of emboli

to the brain which bring about cerebral hemorrhage and sudden death.

Anomalies in the position of vessels may be the cause of death when a therapeutic procedure ordinarily considered to be without risk is carried out. We have encountered only one such case. In order that therapeutic serum might be introduced in the most efficacious manner, it was injected into the cisterna magna of a four-year-old boy (A-36-136) dying of meningitis caused by *Hemophilus influenzae*. This was performed successfully, but when the needle was withdrawn the patient suddenly died. At postmortem examination a massive recent hemorrhage was found over the cerebellum and brain stem. On careful dissection an anomalous branch of the middle cerebral artery was found. This crossed the site of the usual position for cisternal puncture, and had been perforated by the needle.

THE OCCURRENCE OF DEATH AS AN UNEXPECTED TERMINATION OF KNOWN DISEASE

Death sometimes occurs unexpectedly in individuals known to be ill, but not considered to be moribund. Cases of this type are not ordinarily of official interest to the medical examiner, particularly when the patients have been under adequate medical supervision, but similar events may take place in individuals who have not recently been under medical care. This group may be considered examples of uncommon complications of disease processes. A few examples will be given. A three-year-old girl (A-32-92) entered the hospital because of severe dyspnea five days after she developed a cough. A severe laryngotracheobronchitis was discovered, and she was treated over a period of fifty-four days by a variety of methods, including bronchoscopy. Approximately one month before death she began to improve, and this improvement continued until just before death. It was expected that she would leave the hospital in a few days. A tracheotomy tube had been inserted at the time of admission, and was still in place. About four o'clock in the morning on the fifty-fourth day of the illness, the tracheotomy tube was manipulated for the purpose of cleaning it, as had been done many times on previous occasions. A few minutes later the patient suddenly became cyanotic. Respirations then ceased, although the heart continued to beat for a short time. At autopsy the acute laryngotracheobronchitis had disappeared. There was repair of the mucosa throughout the upper respiratory tract. Only a small amount of chronic inflammatory infiltration was found in the submucosa of the larynx, trachea and bronchi and in the peribronchial regions in the lungs. The immediate cause of death was severe emphysema of the mediastinum. The air had entered the mediastinum through an unsuspected sinus tract in the wall of the trachea immediately adjacent to the tracheotomy tube.

We have observed a few cases of an uncommon termination of acute leukemia. A seven-year-old boy (A-36-106) was admitted because of nosebleeds of three weeks' duration. From physical examination and laboratory studies a diagnosis of acute leukemia was made. The patient ran an irregular febrile course during his entire stay in the hospital. He continued to have nosebleeds. The white-cell count rose to 350,000. The respirations became slower and deeper, and the boy died on the seventeenth day. At autopsy the characteristic features of acute leukemia were found. In addition, massive hemorrhage into the cerebellum, pons and medulla and into portions of the cerebrum was present. Thrombosis of vessels with secondary hemorrhage is one of the complications of leukemia and may terminate life sooner than is customary in this uniformly fatal disease.

A few other possibilities may be of interest. It is well known that individuals suffering from brain tumor may die unexpectedly because of sudden enlargement of the mass brought about by necrosis and hemorrhage, sometimes when the existence of a tumor has not been suspected. This is particularly true when a tumor located beneath the tentorium causes sudden obstruction to the flow of cerebrospinal fluid and a rapid increase in intracranial pressure. We have had occasion to perform postmortem examinations on such patients. Thrombosis of the dural venous sinuses may be found as the cause of cerebral hemorrhage and death in infants suffering from eczema, acute or chronic nutritional disturbance and other conditions. On two occasions we have seen hemopericardium secondary to the rupture of an abscess of the myocardium as the cause of an unusual and sudden termination of a bacteremia due to *Staphylococcus aureus*.

Death may occur unexpectedly in infants and children whose past histories contain data which were considered insignificant. A girl (A-32-24) of six months had had feeding difficulty at two and a half months of age and at intervals thereafter. On two consecutive evenings the patient was given emergency hospital treatment because of severe respiratory difficulty. On each occasion on arrival the patient appeared normal. The third evening a similar episode of respiratory difficulty occurred and the patient died within a few minutes after admission. The larynx, trachea and major bronchi were plugged with coagulated

milk. A very small amount of inflammatory change was found in the alveolar walls of the lungs, but no true bronchopneumonia was present.

A recent postmortem examination revealed a surprising finding. A baby (A-37-158) four months of age had had an upper respiratory infection for about one week. It was not considered ill enough to require medical attention. The baby was given by mouth one teaspoonful of camphorated oil (containing approximately 12 to 15 gr. of camphor) by error instead of castor oil. Immediate medical assistance was obtained, and repeated gastric lavage was instituted. Because convulsions occurred the baby was rushed to a hospital, where supportive treatment was given. The clinical picture was considered consistent with a diagnosis of camphor poisoning. The baby died about two hours after admission. Dr. William J. Brickley, who has kindly permitted me to report the findings here, performed an autopsy and discovered an early meningitis caused by *H. influenzae*. Purulent material was found in the meninges over the convexity and over the base of the brain. There was otitis media with extension of the inflammatory process into the mastoid cells. A left lateral sinus thrombosis of several days' duration was also found. Unexpected death early in the course of acute meningitis of several bacterial types is uncommon, but occurs frequently enough for this condition to be kept in mind as a possible cause. In this case the history of ingestion of camphor obscured the clinical picture. A dose of 12 to 15 gr. of camphor is not considered a lethal dose. Had a postmortem examination not been performed the diagnosis would undoubtedly have been camphor poisoning.

UNEXPECTED DEATH IN INFANTS AND CHILDREN PREVIOUSLY IN GOOD HEALTH

We may separate at once from this group a fairly high percentage of cases in which the history was clearly inaccurate. Pneumonia or acute meningitis of several days' duration, repeated aspiration of large amounts of cod-liver oil and similar conditions were found as adequate causes of death. In a second large group of cases death was caused by fulminating infections due to *Streptococcus hemolyticus*, the meningococcus, the dysentery bacillus or other pathogenic bacteria. A detailed discussion of this form of infection has been given elsewhere.[1] In such cases there may be hyperpyrexia and prostration of from two to twenty-four hours' duration, or the patient may be found dead in bed. Severe cerebral edema is usually present, and edema of the optic nerves and a pressure cone are commonly found. Less marked but nevertheless definite evidences of bacteremia are present in the lungs, kidneys, spleen and lymph nodes, and if proper technic is used the offending organism may be recovered from the blood stream. It is possible in some cases to obtain a history of otitis media or an upper respiratory infection on careful interrogation of the parents after the postmortem examination.

UNEXPECTED DEATH IN THE NEONATAL PERIOD

Death during the neonatal period is not usually unexpected if the infant has been under careful medical supervision, although occasional cases of unpredicted death do occur. In general there may be found lesions of the central nervous system such as cerebral hemorrhage, either traumatic or asphyxial in nature, aspiration of amniotic sac contents into the lungs, bronchopneumonia not detected clinically, pulmonary hemorrhage secondary to erythroblastosis fetalis or hemorrhagic disease of the newborn, severe malformations of the heart, massive adrenal hemorrhage, or hemoperitoneum secondary to a rupture of a large subcapsular hematoma of the liver. Atelectasis is not a sufficient cause of death in this period; the cause of persistent unexpanded lungs must be found in each case. It must be remembered that the lungs do not expand fully with the first few breaths, and that a gradually decreasing amount of atelectasis is normal in the neonatal period. In some cases of sudden death in infants, including a few under hospital supervision, the cause of death was found in the lungs. There, lining the alveolar ducts and all available air spaces, were membranes composed of vernix caseosa which had been aspirated in utero. In these patients vigorous breathing for one or two days had resulted in the forcing of the aspired vernix against the alveolar walls, with resultant effective barriers to respiratory exchange within the lungs. Examples in large number might be cited; the causes of unexpected death in the neonatal period cannot be separated from the general pathology of this period. The criteria for reaching a diagnosis of infanticide or stillbirth will be discussed elsewhere.

EXPLANATIONS FOR SUDDEN DEATH WHICH REQUIRE MORE INVESTIGATION

As stated before, in a small number of infants and children who die unexpectedly, postmortem examination including bacteriological and chemical studies may reveal no explanation of death. As experience accumulates, cases may be removed from this unsatisfactory group. Cases of this kind serve as a stimulus for research in this field. Brief mention will be made of a few problems which demand more careful study.

Twice in the last few years we have found on postmortem examination widespread hyperplasia of the islets of Langerhans in the pancreas. Unfortunately no determinations of the blood sugar had been made, but it would appear that hypoglycemia was an important factor in the deaths of these children. Recently Hartmann[2] has published the results of his studies on the blood-sugar values in infants and children, with particular reference to hypoglycemia in patients with massive hemorrhage of the adrenals.

In recent years Waldbott[3] has called attention to the importance of anaphylactic shock as the cause of unexpected death in infants and children. Waldbott has made careful analyses of the histories of some of his patients where data were obtainable. More careful studies on a larger series of patients are indicated before the importance of anaphylactic shock as a cause of unexpected death can be evaluated.

At intervals for the last few years newspapers in Greater Boston have carried reports of babies found dead in bed, strangled by sleeping garments. I have had an opportunity to perform a postmortem examination on only one child concerning whose death that question was raised. I found no evidence of strangulation, but did find a definite early bronchopneumonia. The repeated mention in the newspapers of sleeping garments as the cause of sudden death has caused fear in the lay mind, and has raised an important question in public health which should be answered. It would seem desirable to have complete postmortem examinations performed upon all patients said to have died in this manner.

Further work is necessary also in order to ascertain more exactly the mechanism of death in the small number of individuals who at postmortem examination exhibit some signs of fulminating infection, but not enough to make that diagnosis possible. More careful bacteriological studies, including those for the recognition of viruses, are indicated.

The work of Heymans[4] and his collaborators and of Soma Weiss[5] and his associates has drawn attention to another possibility which may explain certain cases of sudden death at any age. Weiss has shown that some individuals have hyperactive carotid-sinus reflexes, which react on relatively slight stimulation to cause syncope. The application of this possibility to surgical operations in the cervical regions is obvious. I recently examined a child whose death was probably brought about by excessive stimulation of the carotid sinus. The patient, a boy (A-37-95) nearly two years old, had a slow-growing fibrosarcoma in the left cervical region. At operation the tumor lay over and surrounded the bifurcation of the carotid artery on the left. A careful dissection of the tumor mass was carried out during a prolonged operation, at the end of which the blood pressure fell rapidly and the patient died. All data necessary for an unquestionable diagnosis of stimulation of the carotid sinus were unfortunately not obtained. A complete autopsy revealed no organic cause for death, and we are left with either the possibility suggested or a more unsatisfactory one which we might term operative shock. Against the latter possibility is the fact that operations of similar duration performed elsewhere in the body with the same type of technic are frequently carried out without such a termination.

A word must be included concerning the commonest cause for unexpected death as given in city or state mortality records: status thymicolymphaticus. There are few workers who still insist that an enlarged thymus in itself is the cause of death. A number of writers maintain that there is a state, termed "status lymphaticus," which predisposes to unexpected death on slight traumatic insult. This point of view is summarized by Symmers,[6] who states: "As a matter of truth, sudden death in status lymphaticus is a tragedy comparatively rarely enacted, but when it does occur the astonishing rapidity of the blow and the unexpected circumstances in which it is delivered are exceedingly impressive." There are many pathologists, including myself, who believe that there is no justification for the diagnosis of status lymphaticus and that the cause of death must be sought elsewhere in the body. I believe that I may state with fairness that this diagnosis is made all too freely. Certainly here is a problem which will not be closed so long as there are conscientious workers who maintain the existence of a peculiar lymphatic constitution which may be responsible for sudden death. The most pressing problems are those concerned with death under anesthesia or following slight trauma. It may be anticipated that morphologic changes will be absent in such cases. A search must be made, therefore, in other directions. One of the most important objections to the continued use of the diagnosis of status thymicolymphaticus is its implication that the mechanism of death is really understood and that the problem has been solved. A critical survey of the literature on the subject gives no satisfactory explanation based upon acceptable data of either experimental or descriptive nature.

I may suggest one direction of study which may follow in the paths of phenomena now well understood. The experiments of Heymans and of

Hering[7] have demonstrated the existence of important pressoreceptive mechanisms for the reflex regulation of heart rate, blood-pressure, vasomotor tone and respiration. These are located chiefly in the carotid sinuses and the cardioaortic zones. A discussion of this subject may be found in the recent papers of Heymans.[8] It appears worth while to gather data pertinent to the problem at hand concerning the pathologic physiology of these reflex mechanisms, and to conduct a search along similar lines for other reflex zones in the body. This is a suggestion based upon available knowledge, and indicates but one approach to the problem. Whether the explanation for sudden death resides in the lymphatic system or elsewhere in the body, more careful postmortem and experimental studies should be productive of important information in the fields of both physiology and pathology, in addition to clarifying the problem at hand.

SUMMARY

The causes and mechanism of unexpected death in infancy and childhood form a part of the general pathology of those age groups. Complete postmortem examinations reveal the cause of death in most instances. Further study is needed to explain satisfactorily the exact cause or mechanism of death in a small percentage of infants and children who die unexpectedly. In such cases the information obtained from complete postmortem studies will aid in indicating the direction such research should follow.

300 Longwood Avenue.

REFERENCES

1. Farber, S.: Fulminating streptococcus infection in infancy as a cause of sudden death. New Eng. J. Med. **211**:154-159, 1934.
2. Hartmann, A. F., and Jaudon, J. C.: Hypoglycemia. J. Pediat. **11**:1-36, 1937.
3. Waldbott, G. L.: So-called "thymic death," pathologic process in 34 cases. Am. J. Dis. Child. **47**:41-60, 1934.
4. Heymans, C.; Bouckaert, J. J., and Regniers, P.: *Le Sinus Carotidien*. 340 pp. Paris: G. Doin & Co., 1933.
5. Weiss, S., and Baker, J. P.: The carotid sinus reflex in health and disease: its rôle in the causation of fainting and convulsions. Medicine **12**:297-354, 1933.
6. Symmers, D.: Status lymphaticus. Am. J. Surg. **26**:7-14, 1934.
7. Hering, K.: Die klinische Bedeutung der Carotissinusreflexe. Klin. Wchnschr. **6**:232, 1927.
8. Heymans, C.: The Dunham Lectures. New Eng. J. Med. **219**:147-159, 1938.

Discussion

Dr. David C. Dow, Cambridge: What are we going to do in the cases of babies dying without warning? I recently had such a case. You speak of the uncertainty of histories, but the parents of this child were very intelligent people. On the day before death the pediatrician gave the baby a clean bill of health, but by the next afternoon it was dead. The body was sent to the Harvard Medical School, and I received a voluminous postmortem report with a diagnosis of status lymphaticus.

The mention of possible strangulation by sleeping garments reminds me of another fatal case, in which also, I believe, the body was sent to the Harvard Medical School. I was notified by telephone of a diagnosis of status lymphaticus, but with the promise of a later report after further investigation, which I have not yet received.

I have seen other cases, in fact all medical examiners have. We certify the cause of death as status lymphaticus, and this is accepted. We are obliged to file a death certificate within a few days, so that we cannot wait for a laboratory report. What are we to do?

Dr. Farber: I think that Dr. Dow has stated the problem very clearly. In regard to the two cases which he has mentioned, I should hazard a guess that the diagnosis of status lymphaticus has not been made at Harvard within the last fifteen years. I remember the second case very clearly, since I performed the autopsy. I am afraid an incorrect message reached Dr. Dow. It did not come from me, and I regret that our rather voluminous protocol failed to reach the proper hands. Fulminating streptococcal bacteremia was the diagnosis in this case. No evidence of suffocation caused by a sleeping garment was found.

The reason why we hesitate to make a preliminary diagnosis of status lymphaticus is that in every case where that diagnosis has been rendered we have found by gross, microscopic and bacteriological examination adequate explanation for death from another cause. The work of Hammar[1] and Edith Boyd[2] has given us adequate standards for the size of a normal thymus and the normal amount of lymphoid tissue in a healthy child, and has proved that what is commonly called an enlarged thymus is in reality of normal size. I believe that the reason why pathologists have been guilty in the past of giving an incorrect notion of both the size of the thymus and the normal amount of lymphoid tissue in the normal child is that their figures were obtained from sick children whose lymphoid systems had undergone involution.

There are other reasons why this diagnosis should not be made. The practice of treating so-called enlarged thymuses by x-ray is not founded on good evidence, so far as I have been able to learn. Perhaps the most important argument is that although this diagnosis is a convenient and acceptable one it does not cover the facts, and that further investigation which might lead us nearer to the true nature of causes of unexpected death is thus impeded.

I realize that there is a great deal of disagreement on this subject. It is very difficult to decide that a certain condition does not exist on the basis of negative evidence alone. I should appreciate hearing positive evidence on the subject.

Dr. Myrtelle M. Canavan, Boston: How much does the normal thymus weigh?

Dr. Farber: According to the figures given by Edith Boyd, the mean weight of the thymus varies from 10.6 gm. at birth to 31.1 gm. during the period from ten to fifteen years of age. The mean weight then decreases to about 19 gm. at fifty and 13.4 gm. at sixty to ninety. I have found adequate causes for death in children whose thymuses weighed over 60 gm.

A Member: Dr. Dow said that the pediatrician gave the baby in his first case a clean bill of health. Do you assume that the baby had the fulminating streptococcal bacteremia and that the symptoms went unrecognized?

Dr. Farber: The baby was probably not recognizably ill when the pediatrician examined it, but an infection can spread with startling rapidity in early life. We have had a number of examples of this.

A Member: I wish to take issue with reference to the diagnosis of status lymphaticus. I recall the case of a young lad nine years old who was playing in the town dump and was struck a glancing blow on the side of

the head with a bottle thrown by a ragpicker. An ambulance brought him to the hospital but he was dead on arrival. At autopsy the skull was negative, but there was a very slight lesion on the side of the brain; otherwise the brain was perfectly normal. A diagnosis of status lymphaticus was made. The thymus weighed 65 gm. Such cases occur occasionally in the medical examiner's experience, just often enough to make him wonder what is going on. In this case there was no other abnormality to be found.

DR. FARBER: I appreciate the dilemma of the medical examiner, and I am rather happy that I do not have to meet that type of problem. My point is simply this: there may be a number of mechanisms about which we know little which can bring about sudden death. I mentioned one of them — excessive stimulation of the carotid sinus. Could there not be other reflex mechanisms of the same general nature?

I am always left with a feeling of dissatisfaction after reading reports giving status lymphaticus as the cause of death. I still do not understand what happened, and rather than agree, since I do not have to make a diagnosis, I should much prefer to say that I do not know, but should like to find out. This is putting it very bluntly. I am aware, however, of the problems that you have to face.

A MEMBER: I should like to ask a rather pertinent question. In my own family I had a child who had feeding difficulties. An x-ray photograph showed that the thymus was enlarged. At about that time the tonsils were removed, but not until the thymus had shrunk after x-ray treatment. If, in a child of your own, x-ray examination revealed an enlarged thymus, would you have the tonsils removed without x-ray treatment?

DR. FARBER: I am certain that I should not have an x-ray photograph taken at all. At the Children's Hospital in the last twelve years or more no chest photograph has been taken before operation to see whether the thymus was enlarged, yet the death record there is no worse than that of other hospitals where routine plates are taken.

I believe there are very few people today who still believe that an enlarged thymus, as such, is capable of causing damage. Symmers,[3] in a paper published a few years ago, stated that he did not believe that pressure from such a thymus could cause damage. He did, however, concur in the conception of an abnormal lymphatic state as a whole, with the thymus as merely one part of it.

A MEMBER: How does the escape of air into the mediastinum cause death?

DR. FARBER: I should hazard the guess that the sudden collection of air in the mediastinum of a child causes death by a reflex nervous mechanism, perhaps through action on the vagus nerve. I have no detailed information on this subject. Certainly the rapidity of death after such an occurrence points to a nervous phenomenon.

REFERENCES

1. Hammar, J. A.: Fünfzig Jahre Thymusforschung. Ergebn. d. Anat. u. Entwcklngsgesch. **19**:1-274, 1909.
2. Boyd, E.: The size of the thymus and its x-ray shadow in relation to physiologic disturbances and the routine treatment in vogue. Am. J. Dis. Child. **42**:205, 1931. The weight of the thymus gland in health and in disease. *Ibid.* **43**:1162-1214, 1932.
3. Symmers, D.: Status lymphaticus. Am. J. Surg. **26**:7-14, 1934.

THE TREATMENT OF DANGEROUS REACTIONS TO NOVOCAIN*

SAMUEL GILMAN, M.D.†

BOSTON

CLINICAL cases of dangerous reactions to novocain appear to be of three types. The first and most frequent type presents a picture which is chiefly referable to the central nervous system. The reaction begins with apprehension, followed by excitement, delirium, convulsions and respiratory failure. These symptoms may appear within a variable period after giving the drug and frequently the attack is of long duration. Experimentally in animals subcutaneous injection of a minimal lethal dose of novocain produces this same type of reaction, sometimes called the "convulsive" type.

The second type of dangerous reaction to novocain appears suddenly, often immediately on administration of the drug, and is of short duration. In the mild cases one observes signs referable to the circulatory system. The clinical picture is one of sudden pallor, rapid pulse, fall in blood pressure and fainting. These mild attacks usually pass off if the patient's head is lowered. In the severe cases one observes what seems to be sudden circulatory failure. There is a picture of pallor, fainting and sudden stoppage of the heart with immediate death. There may be a single convulsive spasm, or even no muscular contraction but simply collapse. De Takats[1] states that this type of reaction follows inadvertent intravenous injection of novocain. Knoefel and his co-workers[2] found that intravenous injection in animals is more likely to produce this type of reaction, in which convulsions are absent. They explain the different types of reactions on the basis that the concentration of novocain in the blood stream is the important thing. The brain is sensitive to smaller amounts of local anesthetic than is the heart. However, if the concentration is high enough in the blood stream as in the intravenous injection, the heart, being the first organ reached, is the first to fail.

The third type of dangerous reaction to novo-

*Presented at the Combined Clinical Meeting at the annual meeting of the Massachusetts Medical Society, Boston, June 1, 1938. From the Department of Anesthesia, Beth Israel Hospital, Boston.

†Assistant in surgery (anesthesia), Tufts College Medical School; anesthetist, Beth Israel Hospital, Boston.

cain appears to be allergic in nature. Landsteiner and van der Scheer[3] have demonstrated experimentally that simple chemical substances may produce allergic symptoms and shock. Waldbott[4] has collected records of allergic patients in whom an intravenous injection of such nonproteins as sodium iodide, quinine and urethane and the administration of aspirin produced typical allergic shock, and Henderson[5] has stated that many substances containing the benzene ring may lead to sensitization. If a guinea pig is made sensitive by an injection of horse serum, reinjection of the same material, preferably intravenously, two or three weeks later will produce anaphylactic shock. This fundamental experiment is the basis of the widely recognized manifestations of allergy. In a number of the fatalities reported, the history of sudden death within a few minutes after administration of a small quantity, such as 1 cc. of 2 per cent novocain, to a patient who at the occasion of a previous operation well tolerated a much heavier dosage, is highly suggestive of sensitization by the previous injection. The clinical reports of Waldbott,[4] of Mullen[6] and of Waldron[7] furnish further evidence that sensitization to novocain may be acquired. This point has not yet been sufficiently investigated.

The treatment of these dangerous reactions to novocain should begin with attention to certain details which will prevent in many cases the occurrence of untoward reactions. These preventive details are: proper preparation of solutions; proper total dosage used; addition of adrenalin to the novocain solution; attention to past history of the patient in regard to previous administrations of anesthetics; proper technic for administration of the novocain solution; and the preoperative use of the barbiturates.

The use of distilled water in the preparation of novocain solutions must be condemned because such a solution is markedly hypotonic. A 1 per cent solution of novocain in distilled water will hemolyze red cells and rupture connective tissue and fat cells, resulting in some cases in enough tissue necrosis to cause breakdown of a perfectly aseptic wound. To obtain a practically isotonic solution, normal salt solution should be used to prepare novocain solutions for infiltration purposes. Such solutions in saline are acid in reaction but are promptly buffered in the subcutaneous tissues by the ample amounts of carbonates and phosphates in the tissues which allow the novocain base to exert its action. However, for the injection of nerve trunks or roots it is recommended that a buffer solution such as Hartmann's solution be used as a solvent since there is very little tissue fluid available in these locations to buffer the novocain solution. The best solvent for spinal anesthesia is the patient's own spinal fluid which buffers the novocain-hydrochloride crystals before they enter the subarachnoid space. If the novocain solutions are prepared by the hospital pharmacist and must be autoclaved, it should be remembered that they should be autoclaved no more than fifteen to twenty minutes because oxidation of the novocain occurs after this length of time.

A safe dosage of novocain has never been determined. Bieter[8] in studying a summary of the available literature on lethal doses of the local anesthetics in animals has proposed 430 mg. of novocain as an average safe human dose for infiltration purposes. This agrees with the dose given by Hertzler.[9] For injection into highly vascular areas such as the nose, throat and neck and for spinal anesthesia, it is recommended that this dose be cut in half. This agrees with Lundy's[10] figure of 200 mg. as a maximal safe dose for spinal anesthesia. Danger of overdosage in infiltration anesthesia will be materially decreased if the novocain solution is used in the lowest possible concentrations. Twenty-five hundredths per cent solutions of novocain produce a satisfactory anesthesia by local infiltration and 0.5 per cent solutions should not be exceeded for this purpose. Adrenalin added to the novocain solution produces a prolongation of anesthesia due to a local vasoconstriction, thereby slowing the absorption. As a result, smaller concentrations of the local anesthetic may be used, anesthesia is still greatly prolonged, and the danger of toxic symptoms is greatly reduced. It should be borne in mind, however, that adrenalin should not be used in cases of angina pectoris or thyroid toxicity or in regional anesthesia of the digits.

The amount of adrenalin to be used in the novocain solution for infiltration work, as recommended by de Takats,[1] is three drops of a fresh 1:1000 solution to each 30 cc. of novocain solution. This amount agrees with the study of Bieter[8] on the effect of adrenalin on the duration of human wheal anesthesia, where he found that an adrenalin content of 1:100,000 to 1:200,000 produced the longest duration of anesthesia when 0.2 cc. of a 0.125 per cent solution of novocain was injected into the skin on the flexor side of the forearm. Mayer[11] has suggested that the total safe amount of adrenalin added to novocain solution should not exceed 1 mg., that is 1 cc. of a 1:1000 adrenalin solution.

From a technical standpoint the prevention of intravenous injections of novocain will greatly decrease the incidence of dangerous reactions. It is well to remember that the minimal lethal dose of novocain in animals, given intravenously, is one tenth the amount necessary to kill the animal

if given subcutaneously. The use of blunt needles and frequent aspiration with the plunger of the syringe before injection are the chief technical factors in avoiding intravenous injections.

The preoperative use of barbiturates to prevent toxic effects of cocain and its derivatives has been recommended by a number of writers. The first work which indicated that barbiturates may have some antidotal value was done by a Swedish woman physician, Agda Hofvendahl.[12] She reported the successful use of barbital sodium in cocain intoxication in laboratory animals in 1921. Further experimental work along this line has been chiefly done by Tatum and his co-workers,[13] Knoefel and his associates,[2] Maloney,[14] Martin,[15] and Downs and Eddy.[16] Since intoxication from novocain is not frequent, a very large series of cases would be necessary to prove the value of barbiturates as a prophylactic measure. Definite clinical proof is therefore lacking. Reports by Guttman[17] and by Martin[15] are, however, highly suggestive. A fair clinical statement appears to be that such prophylaxis is of benefit but not always successful (see Case 2). The protection afforded in the first type of dangerous reaction described above, as shown by Tatum and his co-workers,[13] makes desirable use of this preventive measure, which is so easily carried out. Maloney[14] has found that sodium pentobarbital is the most efficient of the barbiturates as an antidote in experimental cocain poisoning in animals. Nobody has shown which barbiturate is most efficient as an antidote in man, but in view of animal experimentation, it would seem desirable clinically to use sodium pentobarbital as prophylactic preliminary medication before using novocain solution for anesthesia. Under this plan of medication, the patient would also be well sedated for the operative procedure by this quick-acting barbiturate.

In spite of the above-mentioned details of prevention, one of the three types of dangerous reactions to novocain may occur. The treatment of the first type of reaction when it occurs is illustrated by 2 cases encountered during the past year in which we were able to resuscitate the patient.

CASE REPORTS

Case 1. A 67-year-old housewife was admitted for a high saphenous vein ligation. Two months previously manipulation of the left shoulder and subdeltoid bursa had been done uneventfully under infiltration anesthesia with 50 cc. of 1 per cent novocain. She was given 1/6 gr. of morphine and 1/200 gr. of scopolamine 1 hour before operation and about 20 cc. of 1 per cent novocain was infiltrated in the region of the saphenous opening. Ligation of the saphenous vein was performed. Twenty minutes after the infiltration of the novocain, as the dressing was being applied, the patient became apprehensive and excited and within 1 or 2 minutes had convulsions. After a few minutes automatic respiration ceased. The blood pressure dropped to 60/40 and the pulse was 40 to 60 and of a vagal type. Treatment consisted in lowering of the head, immediate intravenous infusion of 5 per cent glucose in saline to sustain the circulation, and in the course of the next 45 minutes the intravenous administration of 13 gr. of sodium phenobarbital in divided doses. When automatic respiration ceased, artificial respiration was maintained by oxygen insufflations with the E. and J. Resuscitator through a tracheal catheter and later by placing the patient in the Drinker apparatus. All convulsive movements ceased 1 hour after onset, and automatic respiration returned in 1½ hours. The patient regained consciousness in 6 hours and was discharged in good health on the 7th postoperative day.

Case 2. A 47-year-old salesman was brought to the operating room with a diagnosis of diffuse toxic goiter. He was well sedated by preliminary medication of 6 gr. of sodium pentobarbital by mouth. The proposed operation was subtotal thyroidectomy under local novocain anesthesia. Eighty cubic centimeters of 0.5 per cent novocain was infiltrated in the line of incision and in the deeper layers. About 10 minutes after beginning the injection, and before the incision was made, the patient became restless, alert, sweaty and dyspneic; convulsions then supervened and within a few minutes automatic respiration ceased. The pulse was rapid, weak and at times barely palpable, and the systolic pressure dropped to 50. The same resuscitative procedures were applied as in Case 1 except that in this case 14 gr. of Sodium Amytal was given in divided doses intravenously in the course of 1¼ hours. Convulsive movements ceased after 1¾ hours, and automatic respiration returned after 2 hours. The patient showed signs of awakening 4 hours after the onset of the reaction. Convalescence was uneventful and subtotal thyroidectomy under cyclopropane anesthesia was performed 2 weeks later without untoward effects.

Chemical tests on the novocain solution used in this case were all negative. The concentration was found to be correct as labeled. The crystals of novocain from which the solution was made melted sharply at 153°C. Injections of therapeutic doses of this solution into mice produced no untoward effects.

The treatment of the second type of dangerous reaction to novocain where there is an immediate sudden cardiovascular collapse appears to be less successful. Maloney[14] states that from his experience with animals the use of barbiturates in this type of intoxication is of questionable value. We have therefore no satisfactory treatment for this type of reaction beyond the ordinary methods of supporting the circulation and administering oxygen, and there are no available data to indicate what number of patients will survive. The third type of dangerous reaction, namely that due to hypersensitivity, is even less clearly understood, and no data are available as to treatment.

330 Brookline Avenue.

REFERENCES

1. de Takats, G.: The preparation of local anesthetic solutions. Bull. Am. Coll. Surg. 17:40, 1933.
2. Knoefel, P. K.; Herwick, R. P., and Loevenhart, A. S.: Prevention of acute intoxication from local anesthetics. J. Pharmacol. & Exper. Therap. 39:397-411, 1930.

3. Landsteiner, K., and van der Scheer, J.: On specificity of serological reactions with simple chemical compounds (inhibition reactions). J. Exper. Med. 54:295-305, 1931.
4. Waldbott, G. L.: Allergic shock from local and general anesthetics. Anesth. & Analg. 14:199-204, 1935.
5. Henderson, V. E.: cited by Mullen.[6]
6. Mullen, L. M.: Sensitization to novocaine. Canad. M. A. J. 33:306, 1935.
7. Waldron, G. W.: Hypersensitivity to procaine. Proc. Staff Meet., Mayo Clin. 9:254-256, 1934.
8. Bieter, R. N.: Applied pharmacology of local anesthetics. Am. J. Surg. 34:500-510, 1936.
9. Hertzler, A. E.: *The Technic of Local Anesthesia.* Fifth edition. 292 pp. St. Louis: C. V. Mosby Co., 1933. P. 21.
10. Lundy, J. S.: Conservative use of anesthetics; agents and methods. South. M. J. 29:42-45, 1936.
11. Mayer, E.: Toxic effects following the use of local anesthetics. J. A. M. A. 82:876-885, 1924.
12. Hofvendahl, A.: Zur Bekämpfung der Kokainvergiftung. Monatschr. f. Ohrenh. 55:887-892, 1921.
13. Tatum, A. L.; Atkinson, A. J., and Collins, K. H.: Acute cocaine poisoning, its prophylaxis and treatment in laboratory animals. J. Pharmacol. & Exper. Therap. 26:325-335, 1925.
Tatum, A. L., and Collins, K. H.: Acute cocaine poisoning and its treatment in the monkey (Macacus rhesus). Arch. Int. Med. 38:405-409, 1926.
14. Maloney, A. H.: Barbiturates in local anesthetic toxicity. J. Pharmacol. & Exper. Therap. 52:297-306, 1934.
15. Martin, E. G.: Local anesthesia agents; preventive and emergency treatment of toxicity with especial reference to barbituric acid derivatives. J. A. M. A. 91:555-577, 1928.
16. Downs, A. W., and Eddy, N. B.: Influence of barbital upon cocaine poisoning in rat. J. Pharmacol. & Exper. Therap. 45:383-387, 1932.
17. Guttman, M. R.: Acute cocaine intoxication; prophylaxis and treatment with phenobarbital. J. A. M. A. 90:753-755, 1928.

PRACTICAL ASPECTS OF CHILD GUIDANCE*

A Critical Analysis of 500 Cases

CALVERT STEIN, M.D.†

PALMER, MASSACHUSETTS

IN A previous paper I[7] presented an interpretation of the principles and applications of mental hygiene in general practice, together with a brief résumé of 3 cases from the Springfield Hospital Child Guidance Clinic. The present report attempts an answer to the oft-repeated question "What price child guidance?" by presenting the results of six years of study on 500 children whom I examined at the clinic between October 7, 1931, and July 30, 1937. It was our purpose to discover if possible what effect, if any, our guidance had on the children who came to us for help, and to ascertain possible sources of error and new methods of improvement in service to the community.

Hereditary factors, though frequently inquired about by the layman and painstakingly investigated by our social workers, have not been considered of sufficient frequency and importance to demand their inclusion in this study. If too much emphasis appears to have been made on physical and environmental factors, it is only because these fields are the only modifiable ones, since obviously very little can be done to alter a child's heredity after he is born. For detailed discussion of these factors the student is referred to the recent labors of the Committee for the Investigation of Sterilization of the American Neurological Association,[5] and to the control series in the comparative study[6] of hereditary factors in 1000 institutionalized epileptics and 1115 non-epileptic controls.

A review of the literature reveals few articles on the subject of mental hygiene prior to 1929; and of the numerous books and monographs dealing with child guidance that have since been published, still fewer present any detailed statistical data.[1-4, 10] The present review differs from them in that it is a more complete study of an adequate number of cases, all personally examined and treated by the same psychiatrist, in a single clinic serving a stable metropolitan community.

RÉSUMÉ OF FINDINGS

Sex. Two hundred and ninety-five (57 per cent) of the children were boys and 205 were girls.

Age. One fifth were under five years of age, one third were between five and ten, one fourth were between ten and fifteen and one sixth were fifteen or older (mostly under seventeen).

Table 1. *Problems in Order of Greatest Incidence.*

PROBLEM	NO. OF CASES	PER CENT
School difficulties	104	20.8
Enuresis	80	16.0
Sex misconduct	72	14.4
Speech disorders	71	14.2
Feeding problems	64	12.8
Nervousness	63	12.6
Temper tantrums	56	11.2
Disobedience	50	10.0
Stealing and pilfering	48	9.6
Incorrigibility	38	7.6
Nail-biting	36	7.2
Hyperactivity	34	6.8
Retardation	34	6.8
Daydreaming	31	6.2
Habit spasms	29	5.8
Lying	25	5.0
Stubbornness	25	5.0
Thumb-sucking	25	5.0

Observation. The period of observation varied from one month to five and a half years. In 35 per cent of the cases it was under one year, in 20 per cent from one to two years, in 15 per cent

*Presented at a meeting of the Boston Society of Psychiatry and Neurology, October 21, 1937. From the Monson State Hospital and the Springfield Hospital Child Guidance Clinic.

†Senior physician, Monson State Hospital; psychiatrist, Springfield Hospital Child Guidance Clinic.

from two to three years and in 7 per cent for over three years. Twenty-two per cent of the cases are being continued.

Problems. The problems which occur most frequently are listed in Table 1. Forty-seven per cent were problems of habit training, 27 per cent were problems of education and retardation and at least 70 per cent were those of emotional insecurity. Most of the cases presented more than one problem, so that the above figures necessarily overlap. The nine commonest complaints were: school difficulties, 20.8 per cent; enuresis, 16.0 per cent; sex misconduct, 14.4 per cent; speech disorders, 14.2 per cent; feeding problems, 12.8 per cent; nervousness, 12.6 per cent; temper tantrums, 11.2 per cent; disobedience, 10.0 per cent; and stealing and pilfering, 9.6 per cent.

Sources. The sixteen different sources of reference include health agencies, other clinics, private physicians, schools, social agencies, recreational clubs, parents and the juvenile court. The number of cases referred by physicians rose from 2 in 1933 to 18 in 1936 and to 28 in 1937; 162 children (32.4 per cent) were referred by the clinic to local physicians or other clinics.

Diagnoses. Diagnoses were divided arbitrarily for convenience into six groups, as follows: physical, intellectual, educational, social, emotional and psychiatric (psychobiology and psychopathology). A diagnostic summary of the twenty-eight most frequently occurring conditions of all groups, listed in order of incidence, is presented in Table 2. Incorrigibility (including spoiledness and emotional insecurity) heads the list with 46.0 per cent; 36.2 per cent of the patients were of average intelligence, 31.2 per cent had endocrinopathies, 29.6 per cent were malnourished, 25.6 per cent were of dull or borderline intelligence, and 22.4 per cent had speech defects. Twenty-two and four-tenths per cent had visual defects, 20.8 per cent had tonsillitis and in 19.6 per cent there was enuresis. Eighteen and two-tenths per cent had dental defects, 15.6 per cent were delinquents, 14.2 per cent had superior intelligence and 13.0 per cent were feeble-minded. Eleven and six-tenths per cent had school failures, 11.0 per cent were showing effects of adolescent stress and 9.8 per cent had feeding difficulties. The other twelve conditions showed an incidence of less than 10 per cent.

The diagnosis of "normal child" appeared as follows: physically normal, 18 cases (3.6 per cent); emotionally normal, 4 cases (0.8 per cent); intellectually normal (average or superior), 252 cases (50.4 per cent).

Table 2. *Diagnostic Summary.*

DIAGNOSIS	NO. OF CASES	PER CENT
Incorrigibility	230	46.0
Average intelligence	181	36.2
Endocrinopathies	156	31.2
Malnutrition	148	29.6
Dull or borderline intelligence	128	25.6
Dysarthria	112	22.4
Visual defects	112	22.4
Tonsillitis	104	20.8
Enuresis	98	19.6
Dental defects	91	18.2
Delinquency	78	15.6
Superior intelligence	71	14.2
Feebleminded	65	13.0
School failure	58	11.6
Adolescent stress	55	11.0
Feeding difficulties	49	9.8
Nasal obstruction	46	9.2
Puberty	45	9.0
Dermatitis	41	8.2
Immaturity	40	8.0
Sleeping difficulties	37	7.4
Autoerotism	36	7.2
Lymphadenitis	34	6.8
Pes planus	34	6.8
Rachitis	30	6.0
Phimosis	29	5.8
Vasomotor instability	27	5.4
Constipation	22	4.4
Normal:		
Physically	18	3.6
Emotionally	4	0.8
Intellectually (average or superior)	252	50.4

Table 3. *Etiologic Factors.*

ETIOLOGIC FACTOR		NO. OF CASES	PER CENT
Faulty training and discipline		150	30.0
Intellectual retardation		128	25.6
Parental rejection:		115	23.0
Father	27		
Mother	88		
Rivalry and jealousy		109	21.8
Broken homes		94	18.8
Illness or death of other members of family		76	15.2
Unstable, delinquent or immature parents		73	14.6
Adolescent stress		55	11.0
Poverty and economic stress		43	8.6
Malnutrition		42	8.4
Endocrinopathies		39	7.8
School difficulties		37	7.4
Rachitis		30	6.0
Faulty foster-home placement		29	5.8
Alcoholism:		24	4.8
Father	18		
Mother	5		
Patient	1		
High parental standards		24	4.8
Illegitimacy		22	4.4
Indulgent relatives		22	4.4
Congenital defects		21	4.2
Visual defects		17	3.4
Parental neglect		17	3.4
Psychoneurotic and prepsychotic parents		15	3.0
Overwork		14	2.8
Social and religious conflicts		12	2.4
Tonsillitis		12	2.4
Puberty		11	2.2
Undesirable companions		11	2.2
Only child		10	2.0
Adopted child		7	1.4
Auditory defects		7	1.4
Prematurity		6	1.1
Other factors with incidence of 1 per cent or less:			
Convulsions		5	
Epilepsy		5	
Birth injury		5	
Head trauma		5	
Sex conflicts		5	
Superior intelligence		4	
Dermatitis		3	
Inferiority complex		3	
Measles		3	
Unpopularity		3	
Cardiopathy		3	
Chorea		2	
Bronchitis		2	
Rheumatic fever		2	
Anterior poliomyelitis		1	
Asphyxia neonatorum		1	
Left-handedness		1	
Pertussis		1	
Pes planus		1	
Phimosis		1	
Pyelitis		1	
Smoking		1	
Tuberculosis		1	

Only 32 cases were referred for physical complaints, including chorea, strabismus, convulsions, deafness, endocrinopathy, headache, limping, dysmenorrhea, paralysis, skin rash, vomiting and vulvitis. Yet 468 children (96.4 per cent) were found to have one or more of nearly three score of the various physical conditions which space does not permit us to list. Five children (1.0 per cent) were actually psychotic, 132 (26.4 per cent) were psychoneurotic, and 72 (14.8 per cent) presented other personality disorders.

Etiology. Etiologic factors are listed according to the order of incidence in Table 3. The most important ones were the emotional factors. Thus 30.0 per cent of the cases were the result of faulty training and discipline, of whom one fourth had difficulties directly resulting from intellectual handicaps. There was intellectual retardation in 25.6 per cent. In 23.0 per cent there was ill-disguised parental rejection for such reasons as forced marriage, illegitimacy, desertion, economic stress, familial strife, physical or mental handicaps and the like. There were rivalry and jealousy in 21.8 per cent, broken homes in 18.8 per cent, illness or death of other members of the family in 15.2 per cent and unstable, delinquent or immature parents in 14.6 per cent. Poverty and economic stress were directly responsible in 8.6 per cent of the cases. Other factors included brooding over the illness, death or insanity of a parent, sibling or some other member of the household, a licentious or criminal parent, color (in a white neighborhood), obesity, spectacles, a thoughtless teacher, reading and language handicaps, and scores of other seeming tragedies of childhood. Broken homes, which are conspicuously frequent in cases of delinquency (70 to 80 per cent), occurred in only 18.8 per cent of our cases, as mentioned above.

Malnutrition, which was diagnosed in 29.6 per cent of our children, appeared as an etiologic factor in only 8.4 per cent. Similarly endocrinopathies were diagnosed in 31.2 per cent, but appeared as etiologic factors in only 7.8 per cent. The largest item under malnutrition as an etiologic factor is rachitis, which appeared in 6.0 per cent of the cases (the same as the total diagnoses of rickets). A comparable contrast can be made between diagnoses and etiology in most of the other factors. Thus, visual defects were diagnosed in 22.4 per cent of the cases, but appeared as an etiologic factor in only 3.4 per cent.

Dysarthria was diagnosed in 22.4 per cent. Twenty-seven of these patients (5.4 per cent) were stammerers or stutterers. Conversion from left-handedness was found in only 1 case—a child who also exhibited mirror writing.

Therapy. Therapeutic recommendations are listed in Table 4. Over half the cases (53.4 per cent) required parental guidance—usually for the mother; 32.4 per cent were referred to their own physicians or to other clinics; 27.0 per cent for personal guidance, 22.2 per cent for laboratory studies, including a urinalysis on each of the 98 enuretics (usually made in the presence of the child, as part of the therapy), 21.2 per cent for tonsillectomy, 18.2 per cent for dental care, 18.2 per cent for endocrine therapy* and 15.4 per cent for changes in school curriculum. Eleven and six-tenths per cent were requested to return for re-examination after a specified interval, 10.6 per cent received dietary regimes (other than a routine feeding program for the 49 cases with feeding problems), and 10.2 per cent were given other medication, including cod-liver oil. Nine and four-tenths per cent were referred for speech correction, 7.8 per cent for visual refraction and 7.2 per cent for institutional care.

Table 4. *Therapeutic Recommendations.*

RECOMMENDATION OR TREATMENT			NO. OF CASES	PER CENT
Parental guidance			267	53.4
Referred to local physicians or other clinics			162	32.4
Personal guidance — psychotherapy			135	27.0
Laboratory studies (x-ray, hematology, etc.)*			111	22.2
Tonsillectomy or adenoidectomy*			106	21.2
Dental care*			91	18.2
Endocrine therapy			91	18.2
Changes in school curriculum			77	15.4
Specific rewards for effort			72	14.4
Re-examination after specified period			58	11.6
Vocational guidance			54	10.8
Recreational agencies			53	10.6
Dietary (other than routine for feeding problems)			53	10.6
Other medication (including cod-liver oil)			51	10.2
Speech correction			47	9.4
Visual refraction*			39	7.8
Observation and further study			37	7.4
Institutional care:			36	7.2
For feeble-mindedness		22		
For epilepsy:		11		
For treatment	4			
For diagnosis	7			
Others:		3		
Deaf	2			
Psychotic	1			
Social agencies			34	6.8
Sedatives (including treatment for epilepsy)			29	5.8
Physiotherapy*			26	5.2
Changes of home status			22	4.4
Pets			21	4.2
Play and dramatic therapy			18	3.6
Adoptions approved			9	1.8
Special tutoring			7	1.4
Recommendations deferred			2	0.4
Not treated			12	2.4

*Not included in second heading.

Results. Results were interpreted as objectively as possible from reports of parents, schools, agencies, social workers and the psychiatrist's observations of the child himself whenever these were possible.

Fifty-three and eight-tenths per cent of the 500 children are believed to have improved since first coming to the clinic. Of these, 6.6 per cent had recovered. Since no control study is feasible for this type of service, it is impossible to state how many of them would have improved without our help. The contrast between diagnoses and eti-

ology is instructive on this point. These figures compare favorably with those of other clinics as reported by Stevenson and Smith.[9] Twenty-seven per cent of the children was unimproved, and in 19.2 per cent the results were unknown. Since 83 cases were continued, the 96 cases in which the results are as yet unknown will be materially reduced.

Two thirds of the 404 cases with known results, are believed to have improved since their contact with the clinic. The status of the total personality, rather than of specific complaints, formed the basis for objective conclusions as to improvement.

The fact that the percentage of improvement among the girls was not greater, but actually a little less than that among the boys, indicates that we obtained some measure of success in the objective interpretation of their problems, since, according to Russell,[11] the usual story, in public schools for example, is that boys fail three times as frequently as girls.

Co-operation. Co-operation was considered to be satisfactory in 31.6 per cent of the cases. In 13.6 per cent further study and help were declined, usually by the parents. Distressingly high in both co-operative and unco-operative groups, but especially in the latter, was the incidence of broken appointments and failure to reply to follow-up letters. This amounted to 24.2 per cent for the entire study. It was nearly 28 per cent prior to September, 1936, but dropped to 7 per cent after that time, when parents were required to make application during a personal interview with the psychiatric social worker at the clinic. A tendency on the part of some of the more paternalistic referring agencies, in their zeal for securing help for the child, to put pressure on the parents in referring cases, together with a lack of understanding of the purpose and functions of a child-guidance clinic, were important causes, and their removal has already produced excellent results, both in simplifying the therapeutic program and lightening the load of unco-operative cases.

CONCLUSIONS

A statistical analysis of clinical work is an expensive and tedious task; but, like auditing and accounting in industry, it is likely to prove indispensable.

The standardization of diagnoses and improvement of service, by the eradication of waste and the elimination of errors of both omission and commission, are some of the results that help to improve the clinic, which disciplines itself by the laborious task of keeping adequate records and of analyzing them from time to time.

This task can be simplified materially by incorporating a statistical survey form in the face sheet of every record, and completing it before closing the case. The use of code numbers and letters for each problem, diagnosis and etiological factor, and similar items on this sheet which can be duplicated and filed separately for future analyses, should prove helpful.

The physical examinations of these children indicate a very high proportion of removable defects; this indicates that the role of pediatrics in the general practice of medicine is no less important than that of mental hygiene.

Since a large part of the work of a child-guidance clinic lies with the parents, child guidance is starting at the wrong end of the problem. The task could be simplified if the elements of mental hygiene, like those of physical hygiene, were carried into the public schools, and especially into the high schools and teachers' colleges. When more people learn to recognize the importance of emotional insecurity in childhood, prophylaxis will begin to become universally applied.

REFERENCES

1. Branham, V. C.: Analysis of 1671 cases brought to the child guidance clinics of the New York State Department of Mental Hygiene. Psychiatric Quart. 3:569-589, 1929.
2. Kanner, L.: *Child Psychiatry.* 527 pp. Springfield, Illinois: Charles C Thomas, 1935.
3. *Idem:* Problem children growing up. Am. J. Psychiat. **94**:691-699, 1937.
4. Lee, P. R., and Kenworthy, M. E.: *Mental Hygiene and Social Work.* 309 pp. New York: Commonwealth Fund, 1929. Pp. 276-309.
5. Myerson, A., et al: *Eugenical Sterilization: A reorientation of the problem.* 211 pp. New York: The Macmillan Company, 1936.
6. Stein, C.: Hereditary factors in epilepsy: a comparative study of 1,000 institutionalized epileptics and 1,115 non-epileptic controls. Am. J. Psychiat. **12**:989-1037, 1933.
7. *Idem:* The rôle of mental hygiene in general practice. New Eng. J. Med. **214**:665-671, 1936.
8. *Idem:* Studies in endocrine therapy in epilepsy. Am. J. Psychiat. **13**:739-760, 1934.
9. Stevenson, G. S., and Smith, G.: *Child Guidance Clinics: A quarter century of development.* 186 pp. New York: Commonwealth Fund, 1934. P. 149.
10. Thom, D. A., and Winslow, D. C.: A follow-up study of fifty problem children with poor prognoses and fifty problem children with good prognoses. Bull. Mass. Dept. Ment. Dis. **19**:24-37, 1935.
11. Russell, C.: personal communication.

VERMONT STATE MEDICAL SOCIETY

HOUSE OF DELEGATES

THE opening session of the House of Delegates of the Vermont State Medical Society was called to order by the president, Dr. F. J. Lawliss, Wednesday, October 5, 1938, at 8:00 p. m. in the Banquet Room of the Hotel Vermont, Burlington.

President Lawliss appointed as the Credentials Committee:

Dr. F. J. Hurley, Bennington
Dr. T. E. Kirk, St. Albans
Dr. H. E. Upton, Burlington

He requested the members to present their credentials to this committee. The Credentials Committee reported 28 members present. The secretary, Dr. B. F. Cook, called the roll. Those present were as follows:

Addison County	L. S. Walker, Middlebury V. W. Waterman, Vergennes
Bennington County	F. J. Hurley, Bennington
Chittenden County	C. A. Ravey, Burlington H. E. Upton, Burlington H. A. Durfee, Burlington E. H. Buttles, Burlington E. D. McSweeney, Burlington A. B. Soule, Burlington
Franklin County	H. D. Hinman, Enosburg Falls T. E. Kirk, St. Albans C. G. Abell, Enosburg Falls
Lamoille County	B. L. Emerson, Johnson
Northeastern County	E. M. Cleasby, Orleans F. E. Farmer, St. Johnsbury W. B. Fitch, St. Johnsbury W. H. McBride, Island Pond C. G. Schurman, Newport
Rutland County	H. L. Frost, Pittsford R. H. Seeley, Rutland E. J. Quinn, Castleton
Washington County	R. E. Avery, Barre J. L. P. Forest, Waterbury F. S. Kent, Burlington A. T. Marshall, Randolph J. C. O'Neil, Waterbury
Windham County	F. L. Osgood, Saxtons River
Windsor County	M. D. Gardner, Windsor Wayne Griffith, Chester

The report of the Secretary was read, and Dr. R. H. Seeley, Rutland, moved that it be accepted and placed on file; this was seconded and so voted.

Mrs. A. M. Cram's report on the organization of a women's auxiliary to the Vermont State Medical Society was read, and the consensus was that there was not enough interest shown to continue it.

A model constitution and blanks for use by all component county societies, as prepared by the Secretary in conjunction with the Legal Department of the American Medical Association and the legal adviser of the Society, was presented. Dr. F. L. Osgood, Saxtons River, made a motion that the constitution, as presented, be read by a committee of three, and a report made to the Secretary not later than November 1, 1938; this was seconded and so voted. President Lawliss appointed the following committee:

Dr. E. D. McSweeney, Burlington
Dr. E. A. Hyatt, St. Albans
Dr. E. H. Buttles, Burlington

Dr. H. L. Frost, Pittsford, moved that the constitution and by-laws of the Vermont State Medical Society be repealed, and that a committee of three be appointed by the chair to draft a new constitution and by-laws, said committee to report to the House of Delegates in 1939. The motion was seconded and so passed. President Lawliss appointed:

Dr. E. D. McSweeney, Burlington
Dr. E. A. Hyatt, St. Albans
Dr. E. H. Buttles, Burlington

Dr. H. A. Durfee, Burlington, moved that a committee be formed, consisting of the president and secretary of the House of Delegates, Dr. E. H. Buttles, Dr. H. A. Durfee, and any other interested members to meet immediately after adjournment to discuss policies for future spring medical meetings. The motion was seconded and so voted.

Dr. Edward Quinn, Castleton, moved that a committee of two be appointed by the chair to serve as a Library Committee, the duty of which would be to keep a record and inventory of the books and records of the Vermont State Medical Society now housed in the Library of the University of Vermont School of Medicine, and to report to the annual meeting. The chair appointed the following:

Dr. P. D. Clark, Burlington
Dr. P. K. French, Burlington

Dr. H. A. Durfee, Burlington, moved that the Vermont State Medical Society establish a standing committee on maternal and child health. This was seconded and so passed.

Mrs. Carpenter from the Vermont Association for Crippled Children was given permission to address the House of Delegates. She spoke briefly of the functions of the organization. Dr. A. B. Soule, Burlington, moved that the Legislative Committee of the Vermont State Medical Society cooperate in every way possible with the Legislative Committee of the Vermont Association for Crippled Children. The motion was seconded and so passed.

Dr. C. G. Schurman, Newport, moved that the 1939 meeting be held in Burlington, with the Hotel Vermont as headquarters. This was seconded and so voted.

Dr. A. B. Soule, Burlington, presented and read the following resolution from Dr. Clarence Ball, chairman of the Cancer Committee, and made a motion that it be approved and that the work of the Cancer Committee be continued:

WHEREAS, The State of Vermont has no program for cancer control and has one of the highest death rates from cancer; and

WHEREAS, A state survey of the cancer problem has been made at the request of the Vermont State Medical Society by the American Society for the Control of Cancer; be it therefore

RESOLVED, That the recommendations of the Cancer Committee, as tabulated below, be ratified and adopted by the House of Delegates of the Vermont State Medical Society.

RECOMMENDATIONS

1. That the Vermont State Medical Society request the State Legislature to appropriate twenty thousand dollars ($20,000) to develop a cancer program in the State.

2. That the Vermont State Medical Society empower its Cancer Committee with the authority to organize and supervise diagnostic and treatment centers in the several parts of the State, and develop proper statistical records and follow-up systems.

3. That the Vermont State Medical Society develop a program for medical education, using the diagnostic centers to demonstrate the clinical findings in early cancer.

4. That the Vermont State Medical Society continue its interest in and support of the Women's Field Army in its lay educational work.

The motion was seconded and so voted.

Dr. H. E. Upton, Burlington, moved that the Vermont State Medical Society go on record as favoring pre-marital medical examinations. The motion was seconded and so voted.

Dr. C. G. Abell, Enosburg Falls, moved that a committee be instructed to prepare a special program for the annual meeting in 1939 in observance of the one hundred and twenty-fifth anniversary of the founding of the Society. The motion was seconded and so passed.

A brief summary of the activities of the Public Relations Committee was presented by Dr. E. A. Hyatt, St. Albans.

Dr. A. B. Soule, Burlington, moved that the by-laws of the Society be amended to provide for a salary of one hundred dollars ($100) per annum for the Treasurer. This motion was seconded and so voted.

Dr. E. J. Quinn, Castleton, made a motion that the Survey on the Adequacy of Medical Care, as requested by the American Medical Association, be tabled. The motion was seconded and so voted.

Dr. R. E. Avery, Barre, moved that the House of Delegates instruct the Legislative Committee to promote the passage of a law in the next State Legislature, making it obligatory for a person to be a citizen of the United States before he or she can be granted a license to practice any of the healing arts in the State of Vermont. The motion was seconded and so voted.

Dr. W. B. Fitch, St. Johnsbury, nominated Dr. E. A. Hyatt, St. Albans, to serve as president of the Society for 1939. This was seconded and so passed.

For secretary of the Society, Dr. A. B. Soule nominated Dr. F. B. Cook, Rutland. This was seconded and so voted.

It was then moved that the chair appoint a committee to bring in nominations for the remaining officers and committees for the Society and for officers of the House of Delegates at the next meeting of the House on Thursday, October 6. The motion was seconded and so voted.

The chair appointed the Nominating Committee as follows:

Dr. A. B. Soule, Burlington
Dr. W. B. Fitch, St. Johnsbury
Dr. R. H. Seeley, Rutland

Dr. M. D. Gardner, Windsor, moved that the House of Delegates adjourn until 1:00 p. m. on Thursday, October 6. The motion was seconded and passed, and the House adjourned.

The adjourned session of the House of Delegates of the Vermont State Medical Society was called to order by President F. J. Lawliss at 1:30 p. m. Thursday, October 6, 1938, in the Banquet Room of the Vermont Hotel, Burlington.

The Secretary called the roll; 14 members were present.

Dr. A. B. Soule, Burlington, chairman of the Nominating Committee, submitted the following report:

The Nominating Committee presents the following nominees for the consideration of the House of Delegates.

VICE-PRESIDENT — Dr. A. C. Black, Brattleboro

TREASURER — Dr. David Marvin, Essex Junction

AUDITOR — Dr. P. P. Lawlor, Burlington

COUNCILORS

1st District — Dr. L. E. Sample, St. Albans
2d District — Dr. R. E. Seeley, Rutland
3d District — Dr. F. E. Farmer, St. Johnsbury
4th District — Dr. A. M. Cram, Bridgewater

CANCER COMMITTEE

Dr. C. F. Ball, Rutland
Dr. W. W. Angell, Randolph
Dr. J. H. Woodruff, Barre

EXECUTIVE COMMITTEE

Dr. B. F. Cook, Rutland
Dr. C. H. Beecher, Burlington
Dr. A. B. Soule, Jr., Burlington

HEALTH AND PUBLIC INSTRUCTION COMMITTEE

Dr. W. W. Angell, Randolph
Dr. P. D. Clark, Burlington
Dr. C. F. Ball, Rutland
Dr. Louis Rabinowitz, Pittsford
Dr. J. H. Woodruff, Barre

LEGISLATIVE COMMITTEE

Dr. C. H. Beecher, Burlington
Dr. E. D. McSweeney, Burlington
Dr. E. A. Tobin, North Bennington

MATERNAL AND CHILD HEALTH COMMITTEE

Dr. H. A. Durfee, Burlington
Dr. R. E. Corley, Burlington
Dr. E. M. Perkins, Rutland
Dr. R. E. McSweeney, Brattleboro
Dr. Emily T. Wilson, St. Johnsbury

MEDICAL EDUCATION COMMITTEE

Dr. Lyman Allen, Burlington (3 yr.)
Dr. E. H. Buttles, Burlington (2 yr.)
Dr. C. G. Abell, Enosburg Falls (1 yr.)

NECROLOGY COMMITTEE

Dr. C. F. Dalton, Burlington
Dr. G. G. Marshall, Rutland

PUBLICATIONS COMMITTEE

Dr. B. F. Cook, Rutland
Dr. H. E. Upton, Burlington
Dr. Donald Coburn, St. Johnsbury

PUBLIC RELATIONS COMMITTEE

Dr. Stewart Ross, Rutland (3 yr.)
Dr. E. A. Hyatt, St. Albans (2 yr.)
Dr. F. E. Farmer, St. Johnsbury (1 yr.)

DELEGATES

Maine — Dr. W. H. McBride, Island Pond
New Hampshire — Dr. Wayne Griffith, Chester
Massachusetts — Dr. Philip Wheeler, Brattleboro
Rhode Island — Dr. M. D. Gardner, Windsor
Connecticut — Dr. John Trotter, Bennington
New York — Dr. G. G. Marshall, Rutland

AMERICAN MEDICAL ASSOCIATION

Delegate — Dr. B. F. Cook, Rutland
Alternate — Dr. C. G. Abell, Enosburg Falls

ANNIVERSARY CHAIRMAN — Dr. H. A. Durfee, Burlington

OFFICERS OF THE HOUSE OF DELEGATES

President — Dr. F. J. Lawliss, Richford
First Vice-President — Dr. E. D. McSweeney, Burlington
Second Vice-President — Dr. Philip Wheeler, Brattleboro
Secretary — Dr. B. F. Cook, Rutland

RECOMMENDATIONS FOR THE STATE BOARD OF MEDICAL REGISTRATION (TO REPLACE DR. F. J. HURLEY)

Dr. F. J. Hurley, Bennington
Dr. E. J. Quinn, Castleton

Dr. F. E. Farmer, St. Johnsbury, moved that the report of the Nominating Committee be accepted as presented. The motion was seconded and so voted.

Dr. W. B. Fitch, St. Johnsbury, made a motion that the secretary of the Vermont State Medical Society arrange, at the expense of the Society, an annual meeting of the presidents and secretaries of all component county medical societies, and that this meeting be for the purpose of instructing said presidents and secretaries in the rules and general working principles of the Vermont State Medical Society and the county societies. This was seconded and so passed.

Dr. W. B. Fitch, St. Johnsbury, moved that the meeting adjourn. The motion was seconded and so voted. The meeting was adjourned.

ERRATUM

The following equation, appearing on page 512 of the current volume of the *Journal*, in a paper "The Maximum Temperature of Expired Air as a Rapid Measure of Human Body Temperature," by Benedict, Benedict, Lee and Lee,

$$1 \text{ mm.} = \frac{5.90}{124} = 0.48^{\circ}\text{F.}$$

should read

$$1 \text{ mm.} = \frac{5.90}{124} = 0.048^{\circ}\text{F.}$$

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 24471

PRESENTATION OF CASE

A fifty-year-old married American clerk was admitted complaining of progressive weakness of four weeks' duration.

About nine months before entry he began feeling nervous and below par. Two months later his fatigue was such that he was forced to stop work for seven weeks. He was so weak that walking was difficult. After he had returned to work for three weeks he found that he could not carry out his duties and returned home to rest. He felt poorly and had continuous anorexia. Four months before entry he developed fleeting joint pains and aching muscles, mainly in the upper extremities. Small red-black papules appeared on both feet, which were very tender and painful. The feet became markedly swollen, the swelling involving all the toes, but there was no redness or heat. A "rash" which extended from both ankles up to the thighs and consisted of small "bloody" areas about the size of a fingernail appeared at the same time. These spots were not tender and did not itch or bleed. A butterfly rash, which was erythematous and maculopapular, also appeared on the nose and cheeks. Five weeks prior to admission constipation was first noted associated with intermittent upper abdominal pain. One week later he suddenly became "unconscious" for three hours, and his wife could not rouse him. There were no accompanying symptoms or neurological signs. He was very weak and sick for the following week, but at that time the skin rash on his face and extremities, the swelling of the feet and the joint pains quite suddenly disappeared. For the two weeks before entry he was quite clear mentally, and his appetite improved markedly. His weakness persisted. During the year before entry he had dropped from 210 to 130 pounds in weight. He had had no chest pain, palpitation, dyspnea, orthopnea, cough, sputum, hemoptysis or night sweats.

He had had "rheumatism" at the age of sixteen, with chorea and joint pains, and was very sick in a hospital for two weeks. However, a secondary diagnosis of typhoid fever was made.

Physical examination revealed a pale, chronically ill, poorly nourished man with evidence of considerable weight loss. There was a healing erythematous rash of butterfly distribution on the nose and cheeks. His teeth were carious, with obvious pyorrhea. The heart size seemed to be at the upper limit of normal. There was a rough systolic murmur, loudest at the apex and heard also over the aortic area. A short aortic diastolic murmur was also present. A_2 was greater than P_2. A Corrigan pulse was present, with capillary pulsation in the nailbeds. The blood pressure was 115 systolic, 55 diastolic. The lungs were clear. Tenderness was noted over the upper abdomen; there was no real spasm, but the abdominal wall was held tense and palpation was difficult. No masses were palpated though the liver seemed large to percussion. The fingers suggested early clubbing. Rectal examinations were negative.

The rectal temperature was 101.5°F., the pulse 105. The respirations were 24.

Examination of the urine showed a specific gravity of 1.008, a slight trace of albumin and 3 to 4 red cells and 1 to 4 white cells per high-power field. The blood showed a red-cell count of 3,350,000 with 60 per cent hemoglobin, and a white-cell count of 7900 with 85 per cent polymorphonuclears. The red cells were hypochromic and microcytic. The serum nonprotein nitrogen was 28 mg. per cent, the protein 6.6 gm. The sedimentation rate was 1.5 mm. per minute, the hematocrit 27 per cent. The blood Hinton test was negative. The sputum was mucoid and negative for tubercle bacilli. An electrocardiogram was normal except for one ventricular premature beat.

X-ray films showed the heart shadow to be increased on both sides. The hilus shadows appeared increased in width. The costophrenic angles were clear.

On the second hospital day there was a faint to-and-fro murmur heard over the midsternum, which might possibly have been due to friction. The spleen was palpated 2 cm. below the costal margin. A blood culture taken on the third hospital day showed *Bacillus subtilis* in one flask, *Staphylococcus albus* in another. On the fifth hospital day a tourniquet test showed the first petechiae in two minutes and was strongly positive in five minutes. That afternoon he developed lower abdominal cramps and passed a large, bulky dark stool. The cramps continued, and he had the desire to defecate. Lower abdominal pain became steady and apparently very severe. The lower abdomen was tender, without spasm, palpable masses or distention. The white-blood-cell count remained at 7000, and the temperature was 101°F. rectally. On the following day he vomited light-brown material, without much nausea. When his atten-

tion was distracted there was no real abdominal spasm or tenderness. A gonococcal complement-fixation test was negative. A blood culture showed *Streptococcus viridans*. On the seventh hospital day the blood showed a white-cell count of 7700 with 92 per cent polymorphonuclears. On the eighth day his abdominal symptoms were much improved. The specific gravity of the urine was 1.020; there was a slight trace of albumin, and 1 to 4 red cells and 1 to 4 white cells, with an occasional granular cast, per high-power field.

On the twelfth day the patient felt well except for weakness. The blood examination showed a red-cell count of 3,000,000 with 50 per cent hemoglobin, and a white-cell count of 5300. The next day a cough was noted, with coryza, hoarseness and thick yellow sputum. A second blood culture showed *Streptococcus viridans*. The rectal temperature was 103°F. On the sixteenth day sulfanilamide therapy was begun. During the next two weeks the patient was quite ill. The red-cell count gradually decreased to 2,600,000, but the white-cell count rose to 12,400. The urine examination was negative. At this time the blood sulfanilamide level was 22.2 mg. per cent. The serum nonprotein nitrogen was 39 mg. per cent. A blood culture showed no growth. Twenty-one days after sulfanilamide therapy was begun (the thirty-sixth hospital day) the white-cell count was 1400, and the red-cell count 2,360,000; the drug was discontinued. His temperature had remained close to 100°F. rectally.

During the next five days the temperature gradually rose to 102°F. On the evening of the forty-first day he was coughing more than usual. His temperature rose to 104.5°F. rectally, the pulse to 140. The blood showed a white-cell count of 14,200. His throat was clear. There were many coarse and fine rales over both lower lung fields, more on the left. Frequent extrasystoles were heard. A high-pitched apical systolic murmur was present, and there was a suggestion of a diastolic murmur, which could not be proved because of the fast rate. He rapidly failed and died within a few hours.

Differential Diagnosis

Dr. William B. Breed: It seems to me after reasonably close study of this case that the fundamental diagnosis is not much in doubt. Barring the butterfly erythema, which I think made the service consider lupus erythematosus quite seriously, the course, including fever, weakness, anemia, splenomegaly, embolic phenomena and heart disease, with aortic-valve involvement and early clubbing of the fingers, taken with the two blood cultures positive for *Streptococcus viridans*, makes it almost impossible not to make a diagnosis of subacute bacterial endocarditis. The period of "unconsciousness" for three hours is of some interest, and suggests that he might have had a small cerebral embolus. The whole story does not suggest a physiologic or chemical situation. Following that he had a remission, a not uncommon feature of this disease.

One can make a number of comments on the disease course itself, but it is my intention today to discuss the treatment that he received rather than the establishment of a diagnosis, which I am prepared to support as subacute bacterial endocarditis. It may be worthwhile first to discuss his death, however, and try to determine what was its immediate cause. There was, during the period of treatment, an effect on the granulocytes from the sulfanilamide, but after the drug was discontinued his white count returned to 14,000. Therefore, I think it quite unlikely that he died of agranulocytosis following sulfanilamide treatment. The most likely cause of his death was of course his disease; but the acute situation could be perfectly well explained on the basis of pulmonary emboli, which presupposes that he had endocarditis in his right heart.

A discussion of sulfanilamide therapy at this point seems timely. I am sure we are all familiar with the attitude which is being taken in the usual cases of hemolytic streptococcal infection of the throat, gonococcal arthritis and infection of the urinary tract with colon bacilli, and I think that we are all convinced that sulfanilamide should not be given unless we have clear-cut indication of infection with an organism which is susceptible to the effects of the drug. However, when we come to this disease, subacute bacterial endocarditis, which is a desperate situation for which we have no cure, it seems justifiable to give these patients at least the benefit of trial even though the green streptococcus is resistant to the drug. I believe there has been no known case of cure with the use of sulfanilamide. I have heard of two cases at the Johns Hopkins Hospital, in which the blood cultures were made sterile for a certain time but, as I have said, there have been no cases reported in which there was a cure over a period of many months. Dr. Edward F. Bland tells me that there is one patient at the House of the Good Samaritan who had a good remission for two months, during which time a blood level of 10 mg. per cent of sulfanilamide was maintained; but while this was being maintained with the use of 120 grains a day, the patient began to go downhill and had recurring symptoms of the disease.

Dr. Charles L. Short had one case in which he maintained the blood level at 22 mg. per cent; the blood culture became sterile and the man went back to work, where he has remained for four months, feeling really very much better; but Dr. Short now reports that the patient is again beginning to have embolic phenomena.

Dr. Charles L. Short: A blood culture taken last week was positive.

Dr. Breed: Dr. Milton J. Quinn and I have had a case in which we gave 200 gr. daily for five days and could only maintain a blood level of 16.7 mg. per cent. There was no immediate effect during that time, either on his temperature or on his symptoms. However, after discharge he felt so much better that he was willing to entertain the suggestion that it be done again. That brings up the question of technic in these desperate cases; whether one should attempt to give huge doses and maintain twice the usual level, namely around 20 mg. per cent, for a few days, or whether one should maintain the level of 10 mg. per cent over a period of weeks or months. I do not believe we have had enough experience to know which technic is the better.

I have asked Dr. William W. Beckman to give me some figures on large doses and high levels of sulfanilamide. One case that he had took 250 gr. daily and established a blood level between 35 and 40 mg. per cent, which he maintained for ten days; this, interestingly enough, had no effect on his gonorrheal urethritis. The patient did not develop agranulocytosis. Another patient of his took 300 gr. daily, establishing a blood level of 33 to 38 mg. per cent, which was maintained for two weeks but had no effect on the gonorrheal urethritis. The patient was irritable and had cerebral symptoms, which I think is a common finding with such high doses. Dr. Quinn's patient certainly had cerebral symptoms. He became lethargic and for hours at a time lost contact with things that were going on, but he could be aroused. I am not at all sure that we can say that the level of sulfanilamide has any correlation with the production of agranulocytosis. Please understand that this is by no means a report. It is rather a general discussion of this most interesting subject which needs much more extensive illumination.

To go back to today's case, I do not see how one could consider another diagnosis very seriously in view of the fact that we have a very good story for subacute bacterial endocarditis and two positive cultures of *Streptococcus viridans*. Therefore I shall offer no alternative diagnosis. Whether he had pulmonary emboli just before his death or just ordinary bronchopneumonia, I have no idea. I should think one would have to choose between those two.

Dr. Tracy B. Mallory: Do you want to tell us which valves you expect to find involved?

Dr. Breed: That the aortic valve was involved is reasonably clear. If one could prove he had pulmonary emboli, then one could assume he had pulmonary-valve involvement. I should say from the clinical story that it is reasonable he might have involvement of both valves, but I am not sure about the pulmonary valve. We must remember that pulmonary-valve involvement is rare.

Dr. Mallory: Do you think he had lupus erythematosus?

Dr. Breed: I do not!

Clinical Discussion

Dr. Wyman Richardson: This patient came in while I was on service. I went over him with the students, and because of the joint symptoms, the rash and the urinary findings we believed that the history was quite suggestive of lupus erythematosus, although the patient was a male and rather old for the disease. I wrote a note saying that the clinical diagnosis of acute lupus disseminatus was likely and that there was underlying rheumatic heart disease, with subacute bacterial endocarditis, which is not uncommon with lupus. That was when he first came in and before we had blood cultures. I do not know what Dr. Mallory found because I went off service a month before the patient died.

It is fair to say that when we looked him over the first time the skin lesion was quite typical of acne rosacea and did not suggest lupus erythematosus. It is my feeling that the latter diagnosis is often a clinical one. I know that Dr. Mallory has difficulty from the pathological point of view in making such a diagnosis, and if one were trying to decide between rheumatic heart disease, which is very definite, and lupus erythematosus, which is very indefinite, I think one would agree with Dr. Breed and stick to the diagnosis of rheumatic heart disease.

One of the things that interested us was the cause of the severe abdominal pain. We thought it might be a mesenteric embolus. I think we decided it probably was, although recovery from a large mesenteric thrombosis is unlikely. I do not know what the final diagnosis was, but I remember that all those who were talking about lupus erythematosus in the beginning finally came around to the idea that the diagnosis was rheumatic heart disease with subacute bacterial endocarditis.

DR. BREED: I should like to make one correction. I am not emphasizing his rheumatic heart disease as such. I suppose he did have rheumatic heart disease with aortic involvement, but that merely was the background for this disease entity of subacute bacterial endocarditis.

DR. RICHARDSON: I did not commit myself as to which valve might be involved.

DR. BREED: That was wise.

CLINICAL DIAGNOSES

Acute disseminated lupus erythematosus.
Subacute bacterial endocarditis.
Rheumatic heart disease, aortic and mitral.
Bronchopneumonia.

DR. BREED'S DIAGNOSES

Subacute bacterial endocarditis (aortic-valve involvement).
Rheumatic heart disease.
Bronchopneumonia?
Pulmonary emboli?

ANATOMICAL DIAGNOSES

Subacute bacterial endocarditis, aortic and mitral.
Endocarditis, rheumatic, healed, aortic and mitral.
Aortic insufficiency.
Infarcts of spleen and kidney.
Hydrothorax, left.
Hydropericardium.
Bronchopneumonia, right base.
Cholelithiasis.

PATHOLOGICAL DISCUSSION

DR. MALLORY: He did have subacute bacterial endocarditis on both the aortic and mitral valves and evidence of old rheumatic involvement on the mitral. The aortic valve was sufficiently destroyed by the bacterial process to make it impossible to determine whether it also had an underlying rheumatic lesion. The vegetations were filled with bacteria, supporting the positive blood cultures. The right side of the heart was negative.

As to whether the patient had acute lupus erythematosus, I cannot say. He showed nothing at autopsy which would be pathognomonic of it and did not show the few things that one might have expected. He did not have polyserositis. He had fluid in the cavities, but it was a clear transudate that came, I should judge, from some degree of heart failure. He had an embolic nephritis of the kind usually seen in bacterial endocarditis but also found in lupus erythematosus.

The bone marrow was quite interesting. It was definitely abnormal. It showed some degree of hyperplasia of the red-cell series, and very marked hypoplasia and lack of differentiation in the white-cell series. The general effect was that of a moderately hypoplastic marrow, which would be unusual in bacterial endocarditis. I am tempted to attribute the changes to the sulfanilamide. I have also seen similar bone marrow in lupus erythematosus, however.

DR. CHESTER M. JONES: What do you consider the diagnostic criteria of lupus erythematosus?

DR. MALLORY: There are none. One is presented with a corpse and the organs appear practically normal. Pericarditis and diffuse enlargement of lymph nodes have been our most constant findings.

DR. JONES: There is one question that has interested me recently. We have noted that an occasional patient with subacute bacterial endocarditis, in spite of an elevated temperature, may have a normal sedimentation rate. This is quite unusual in other forms of infection. I know that I have seen three or four such instances and wonder if it has any diagnostic significance in differentiating subacute bacterial endocarditis and rheumatic heart disease.

DR. EDWARD F. BLAND: We have seen younger people with a sedimentation rate just the other way, that is, much higher than we should expect from the infection. Perhaps the age has something to do with it.

DR. BREED: Were there any pulmonary emboli?

DR. MALLORY: No; he had bronchopneumonia. I did not mention that there were both splenic and renal infarcts. These were the only things we found that might have explained the attack of abdominal pain.

CASE 24472

PRESENTATION OF CASE

A sixty-three-year-old married American mechanic entered complaining of precordial pain.

Two weeks preceding entry the patient was awakened by a sharp pain in the precordial region, which was aggravated by deep breathing and change in position. It subsided shortly after taking bicarbonate of soda. The following day he had a dull precordial ache. An electrocardiogram taken by his physician was normal. Fluoroscopy and x-rays revealed a growth in the left chest the size of a baseball. His pain gradually subsided, but was perceptible on entry. He had no cough, sputum, fever, hemoptysis or weight loss. His past and family histories were noncontributory.

Physical examination showed a healthy appearing man in no distress. He had a few anterior teeth and some dental appliances. The condition of his remaining teeth was only fair, with mild pyorrhea. Examination of the heart was negative. The blood pressure was 130 systolic, 85 diastolic. There were diminished breath sounds and tactile fremitus, and dullness, in the left upper anterior chest. A mass was palpated in the right upper quadrant which descended on inspiration and seemed to be the edge of the liver.

The temperature was 98.6°F., the pulse 102, the respirations 20.

Examination of the urine was negative. The blood showed a red-cell count of 4,260,000 with 75 per cent hemoglobin, and a white-cell count of 7700 with 67 per cent polymorphonuclears. The serum nonprotein was 23 mg. per cent, the protein 7.2 gm. A blood Hinton test was negative.

X-ray films of the chest showed a large, sharply defined, lobulated tumor in the left lung field, extending from the level of the fifth to the ninth rib posteriorly. In the lateral view the mass was seen to lie more anteriorly than posteriorly and to reach the chest wall along the anterior axillary line. The left costophrenic angle was obliterated by a small amount of fluid. The right lung field was clear. There was no evidence of collapse on either side. The heart shadow was not remarkable. An intravenous pyelogram was negative. The bladder showed a pressure defect produced by a moderately enlarged prostate. Films taken after a diagnostic pneumothorax showed air in the pleural cavity apparently surrounding the mass in the left upper lobe. The mass moved with the lung on inspiration and was apparently free except at its upper lateral margin, about the level of the fourth rib posteriorly, where there were probably some adhesions. The mass was within the lung and did not arise from the chest wall.

On the tenth hospital day an operation was performed.

Differential Diagnosis

Dr. Richard H. Sweet: This is an interesting history because of the fact that it is so lacking in detail. We have the sudden onset of precordial pain which makes one think of some acute episode such as coronary thrombosis, but the only significant finding which develops is that he had a tumor in his chest, which would seem to be a most unusual sequence of events.

Because there is no comment as to whether the liver edge was irregular in outline or nodular, we can assume that it was normal. The laboratory findings were negative except for slight anemia.

This x-ray film demonstrates the pneumothorax very well, and the tumor seems to be adherent to the chest wall at one point. The presumption, however, is that it was a pulmonary tumor rather than one arising in the chest wall. I imagine the physician in charge of the patient was surprised by this x-ray finding.

The pain is interesting. I do not associate precordial pain with tumors of this sort. We must therefore discuss the differential diagnosis of a sizable tumor in the lung field that is demonstrable by x-ray and accompanied by practically no symptoms. The physical findings of dullness and diminished breath sounds one would expect.

First of all is this likely to be a benign or a malignant tumor? I have no idea as to how long it was there. The fact that he had so few symptoms, and especially the absence of hemoptysis and cough, suggests that it was probably not a malignant growth. Let us assume, however, for the time being that it was a malignant tumor. Was it carcinomatous? In an examination of the history we have very little in favor of a diagnosis of carcinoma of the lung. Since it was a large, lobulated tumor with a rounded contour, the x-ray appearance is not suggestive of carcinoma. There was no hemoptysis — a characteristic though not essential symptom of carcinoma of the lung. He had only slight anemia, and there are no other symptoms, such as cough or wheezing, to point to disease of the lung except this precordial pain which prompted the doctor to obtain the first x-ray. I should be inclined, therefore, to feel that this was not a primary carcinoma.

Could it be a metastatic carcinoma? That I think is more likely, and apparently he was investigated with that in mind. An intravenous pyelogram was negative. They must have been thinking of hypernephroma. There was nothing to suggest primary malignant disease elsewhere. The prostatic enlargement was presumably benign. The absence of bleeding is a little bit more in favor of metastatic than of primary malignant tumor, but I think we shall have to discard the former as a diagnosis.

One other malignant tumor of the lung that we see not infrequently is lymphoblastoma, but there is a little more to be said for the diagnosis of lymphoblastoma than for that of carcinoma. The relative absence of pulmonary symptoms, the lobulation of the tumor and the x-ray appearance might be more consistent with it than with carcinoma. Sarcoma we see so infrequently and know so little about that I shall only mention it. We need not discuss mediastinal tumors because this was obviously in the lung.

Could it be some form of benign pulmonary

neoplasm? Could it be a cyst? It certainly has no fluid level — no combination of air above and dense shadow below. It looks like a solid tumor. If it is a cyst, it is completely filled with fluid. Its margin is smooth and round, as one might expect with a cyst, but I do not believe that it is one. Echinococcus cyst need not be brought into the picture. What other sort of benign tumor might it be? It is not the so-called adenoma of the lung which usually gives an entirely different picture — bronchial invasion, bleeding and partial or complete obstruction of the bronchus. The only benign tumor of the lung which I can think of that it might possibly be is a chondroma. I cannot see any evidence of calcification in this tumor. Do you see any, Dr. Hampton?

DR. AUBREY O. HAMPTON: No.

DR. SWEET: My inclination is to believe that this is a benign growth, just what type — unless it is chondroma — I am not sure. Lymphoblastoma is a very remote possibility, chiefly because of the fact that the patient had so few symptoms and remained so well in the presence of a large neoplasm in the lung which must have been there for a considerable period of time.

DR. TRACY B. MALLORY: Have you anything to add, Dr. King?

DR. DONALD KING: I am not sure whether I saw him or not. At the present time, if I had to guess, I should say malignant disease. It looks, however, something like the hamartoma we had a while ago.

DR. HAMPTON: It does not show a line of diminished density around it such as we saw in the hamartoma and which is supposed to be due to fat. I wonder if the lobulation means anything. A tumor showing so many lobulations is more likely to be malignant than benign. That is a very lobulated mass.

DR. MALLORY: I think lobulation would be possible in hamartoma.

DR. HAMPTON: Yes, in chondroma too.

DR. EDWARD D. CHURCHILL: We thought it increased in size in the three-week interval between the doctor's note and ours.

CLINICAL DIAGNOSIS

Carcinoma of the lung.

DR. SWEET'S DIAGNOSIS

Chondroma of the lung.

ANATOMICAL DIAGNOSIS

Fibrosarcoma of the lung, probably primary.

PATHOLOGICAL DISCUSSION

DR. MALLORY: The lung was resected by Dr. Churchill and found to contain a very large solid tumor. It extended to the pleural surface, and it was necessary to resect a small portion of a couple of ribs where it was adherent to them. In the other direction it reached down very deeply into the lung. Most of it consisted of a large spherical mass, but there were irregular knob-like projections, one of which was more or less separate from the rest of the tumor. It was hard, white and fibrous. A frozen section, done at the time of operation, showed a spindle-cell sarcoma, and the later sections proved it to be quite typical of a well-differentiated fibrosarcoma. From the pathological examination we cannot rule out the possibility that this is a metastatic tumor. If my recollection is correct we have had one primary benign fibroma within the lung. We have certainly had one primary fibrosarcoma, — a small one evidently starting in a bronchus, — and we have had two other cases where large fibrosarcomas involved the lung but also the pleura, including a good deal of the pleural cavity, so that it was impossible to say with certainty whether they were primary in the lung or in the pleura. In at least three of these cases, however, we are quite confident the tumor started within the lung itself, and I think one has to consider fibromas and fibrosarcomas as possibilities in the differential diagnosis of these pulmonary tumors.

DR. WYMAN RICHARDSON: Was the pericardium involved?

DR. MALLORY: No.

DR. CHURCHILL: It is fair to add that he made a very good convalescence, was discharged at the end of three weeks and is now back at work.

DR. MALLORY: The tumor is not a particularly rapidly growing one, but we had to classify it as malignant. There is a possibility of metastases, but there is also a reasonable chance that he has been cured.

The New England
Journal of Medicine

Formerly the
Boston Medical and Surgical Journal

Established in 1828

Owned by the Massachusetts Medical Society and Published under the Jurisdiction of the Committee on Publications

Official Organ of
The Massachusetts Medical Society
The New Hampshire Medical Society
The Vermont State Medical Society

Editorial Board

George G. Smith, M.D.
Joseph Garland, M.D.
William B. Breed, M.D.
George R. Minot, M.D.
Frank H. Lahey, M.D.
Shields Warren, M.D.
George L. Tobey, Jr., M.D.
C. Guy Lane, M.D.
William A. Rogers, M.D.
Dwight O'Hara, M.D.
John P. Sutherland, M.D.
Stephen Rushmore, M.D.
Hans Zinsser, M.D.
Henry R. Viets, M.D.
Robert M. Green, M.D.
Charles C. Lund, M.D.
John F. Fulton, M.D.
A. Warren Stearns, M.D.

Associate Editors

Thomas H. Lanman, M.D.
Donald Munro, M.D.
Henry Jackson, Jr., M.D.

Walter P. Bowers, M.D., Editor Emeritus
Robert N. Nye, M.D., Managing Editor
Clara D. Davies, Assistant Editor

Subscription Terms: $6.00 per year in advance, postage paid, for the United States; Canada, $7.04 per year; $8.52 per year for all foreign countries belonging to the Postal Union.

Material for early publication should be received not later than noon on Saturday.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to the New England Journal of Medicine, 8 Fenway, Boston, Mass.

POSTGRADUATE ASSEMBLY

The Committee on Postgraduate Instruction of the Massachusetts Medical Society and the various subcommittees in charge of the first New England Postgraduate Assembly deserve the highest praise. In the first place, the carefully planned program so appealed to the physicians of New England that the total attendance — nearly 900 — far exceeded the committee's fondest hopes. Secondly, the arrangements had been so made that, in spite of the large attendance, everything ran along smoothly. Thirdly, the guest speakers so presented their topics that all the talks were received with the greatest enthusiasm. In other words, the assembly was a huge success. One of the guest speakers volunteered the information that the audience was the "best spirited and the most enthusiastic" of any he had ever addressed.

Possibly this type of meeting, that is, one designed primarily for instruction in advances in the diagnosis and treatment of disease by those who speak with authority, has received in New England less attention, as a means of postgraduate instruction, than it deserves. In any event, there is ample justification for the establishment of the New England Postgraduate Assembly as an annual fixture. Even with the relatively brief preliminary notices this year, over 70 physicians registered from Maine, nearly 50 from New Hampshire, over 20 from Vermont, over 30 from Rhode Island and 15 from Connecticut. With official sponsorship by most, if not all, the medical societies of the New England states — which at the moment seems more than likely — the future assemblies will be assured of even greater success than the first.

INCIDENCE AND COST OF SYPHILIS

That syphilis is always present in a community is no longer doubted, but the number of cases per year and the actual costs of the disease are unknown quantities. An article concerning a study of the incidence and costs of syphilis at the Boston City Hospital over a ten-year period and published in this issue of the *Journal* is therefore of particular interest.

Syphilis was found almost uniformly in 2.25 per cent of the annual admissions to the hospital during these ten years, in spite of the fact that the total admissions doubled during this time. These figures were obtained without an intensive examination of patients for the possibility of syphilis and before the routine use of serological tests on all admissions. If such had been done, these figures would probably have been doubled or at least considerably increased.

There is so little material available with regard to the cost of syphilis to a community, either in the actual cost of medical services, drugs and hospitalization or in the economic loss to the individual affected or to the employer, that the figures pertaining thereto should excite the interest of everyone. When a minimum of $70,000

a year is spent in one hospital for the hospitalization of patients with syphilis, — about one third of this amount being for the care of cases of cardiovascular and cerebrospinal syphilis, — one realizes that adequate treatment of the disease, particularly in its later stages, is an expensive item to be reckoned with in any community. In the present era these costs should begin to decrease to a marked degree. With more publicity about syphilis, with better means of detection, with treatment facilities available to everyone and with adequate treatment of early cases and follow-up of delinquents, late manifestations should become much less frequent.

To every thinking physician, and layman as well, these figures place additional emphasis on the need for the early detection of syphilis and adequate treatment, judged by modern standards, as preventive measures of the highest order in eliminating such disability and its incidental expense.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY

RAYMOND S. TITUS, M.D., *Secretary*
330 Dartmouth Street
Boston

PLACENTA ACCRETA

Since September 1 the subject of ruptured uteri has been covered in this column. The condition has been thoroughly illustrated by actual cases. The next condition to be considered is placenta accreta.

Placenta accreta is the abnormal adherence, either in whole or in part, of the afterbirth to the underlying uterine wall. Its pathological background is a complete or partial absence of the decidua basalis and in particular of its deep or spongy layer which lies upon the myometrium. Absence of the decidual coat forces the chorionic villi to attach themselves directly to the muscular coat of the uterus and even in some instances to penetrate deeply into the wall of the organ giving rise to the condition known as placenta increta or when the villi actually perforate the uterine wall, to that called placenta percreta.

A series of selected case histories by members of the section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the section.

The condition is rare in primiparas, there being only 2 recorded cases. In about 40 per cent of cases there is a previous history of manual extraction of a placenta, vigorous curettage or uterine sepsis.

The mortality rate is high — 37.2 per cent — and rises to 66.6 per cent in those cases in which the placenta was extracted manually. No deaths have been reported, however, among those cases in which no attempt was made to extract the placenta and the uterus was removed supravaginally. It is evident, therefore, that once the diagnosis is established, no other method of treatment should be considered. As in all other complications of parturition accompanied by severe hemorrhage, transfusion should be employed early and repeated as many times as necessary.

The fact that cases of placenta accreta are being recognized both clinically and pathologically today when twenty years ago the condition was considered to be a rarity is proof that obstetric pathology is a true specialized branch of pathology. The art of obstetrics will improve with the development of more men interested in obstetric pathology.

The subject will be illustrated by actual cases which will give the clinical characteristics of placenta accreta and demonstrate its proper and improper treatment.

MEDICAL POSTGRADUATE EXTENSION COURSES

The following sessions, given by the Massachusetts Medical Society in co-operation with the Massachusetts Department of Public Health, the United States Public Health Service and the Federal Children's Bureau, have been arranged for the week beginning November 28:

BRISTOL SOUTH (New Bedford Section)

Friday, December 2, at 4:00 p. m., at the St. Luke's Hospital, New Bedford. Subject: The Indications and Contraindications for Removal of Tonsils and Adenoids. Instructor: Warren R. Sisson. Robert H. Goodwin, *Chairman*.

ESSEX NORTH

Friday, December 2, at 4:30 p. m., at the Lawrence General Hospital, Lawrence. Subject: Medical Complications in Pregnancy. Instructor: James C. Janney. John Parr, *Chairman*.

ESSEX SOUTH

Tuesday, November 29, at 4:00 p. m., in the Nurses' Home of the Salem Hospital, Salem. Subject: The Indications and Contraindications for Removal of Tonsils and Adenoids. Instructor: Lyman G. Richards. Walter G. Phippen, *Chairman*.

HAMPSHIRE

Wednesday, November 30, at 4:15 p. m., in the Nurses' Home of the Cooley Dickinson Hospital, Northampton. Subject: Bright's Disease and Hyperten-

sion: Evaluation of New Therapy: Diagnosis. Instructor: Robert S. Palmer. Warren P. Cordes, *Chairman*.

NORFOLK SOUTH

Monday, November 28, at 8:30 p. m., at the Quincy City Hospital, Whitwell Street, Quincy. Subject: Sepsis. Instructor: Thomas R. Goethals. David L. Belding, *Chairman*.

PLYMOUTH

Tuesday, November 29, at 4:00 p. m., in the Rosa Field Nurses' Residence, Brockton Hospital (rear of hospital), Brockton. Subject: Syphilis: Latent Syphilis — Diagnosis and Treatment. Instructor: Rudolph Jacoby. Walter H. Pulsifer, *Chairman*.

WORCESTER

Tuesday, November 29, at 8:30 p. m., in the Nurses' Home of the Milford Hospital, Milford. Subject: Breech Deliveries. Instructor: Raymond S. Titus. Joseph Ashkins, *Chairman*.

WORCESTER NORTH

Friday, December 2, at 4:30 p. m., at the Burbank Hospital, Fitchburg. Subject: Gonorrhea: Modern Treatment of Gonorrhea. Instructor: Oscar F. Cox, Jr. George P. Keaveny, *Chairman*.

DEATHS

CHASE — HEMAN L. CHASE, M.D., of East Alstead, New Hampshire, died November 17. He was in his eightieth year.

Born in Newton, he was graduated from Harvard College and received his degree from the Harvard Medical School in 1887. He served his internship in the Boston City Hospital and later spent a year in the hospitals of Vienna, Prague and other European cities.

Because of his interest in nervous and mental diseases Dr. Chase was selected as volunteer assistant physician on the staff of the Boston Psychopathic Hospital when it was built. He later served on the staff at the Boston State Hospital and was resident physician at the Herbert Hall Hospital, Worcester. At the time of his retirement in 1924 he was on the staff at Danvers State Hospital.

Dr. Chase was a member of the American Medical Association and the Massachusetts Medical Society.

A daughter and a son survive him.

PAPAVASILIOU — VASILIOS K. PAPAVASILIOU, M.D., of 157 Huntington Avenue, Boston, died August 1. He was in his fifty-fifth year.

Dr. Papavasiliou received his degree from the Tufts College Medical School in 1915. He was a member of the American Medical Association and a fellow of the Massachusetts Medical Society.

NEW HAMPSHIRE MEDICAL SOCIETY

DR. HENRY O. SMITH HONORED

On November 10 the townspeople of Hudson gave a surprise dinner for Dr. Henry O. Smith on the fiftieth anniversary of his practice in the community. Over two hundred attended, and speeches were made by representatives of the local hospitals, the School Board and the Board of Selectmen and by Dr. Frank E. Kittredge, of Nashua. In addition to many baskets of flowers, he was presented with a gold watch in memory of the occasion. Dr. Kittredge brought out the fact that the community had received continuous medical service from a "Dr. Smith" since 1850, at which time Dr. David O. Smith, Dr. Smith's father, began to practice in Hudson.

DEATH

NUTE — WILLIAM H. NUTE, M.D., died in Exeter, New Hampshire, on August 18, 1938. Dr. Nute was born in Farmington, New Hampshire, May 8, 1858. He graduated from Bowdoin College Medical School in 1881. He was one of the leaders in founding the Exeter Hospital. He is survived by his widow and one son. He was a member of the New Hampshire Medical Society, the Rockingham County Medical Society and the American Medical Association.

VERMONT STATE MEDICAL SOCIETY

VERMONT DEPARTMENT OF PUBLIC HEALTH

The following communicable diseases were reported to the office of the Department of Public Health during the month of September: whooping cough, 100; measles, 3; mumps, 27; undulant fever, 5; poliomyelitis, 1; chickenpox, 14; diphtheria, 7; German measles, 1; scarlet fever, 23; tuberculosis, 10.

The Laboratory of Hygiene made 2399 examinations, the details of which are:

Examinations	for	diphtheria bacilli	183
"	"	typhoid fever (Widal reaction)	75
"	"	undulant fever	114
"	"	gonococci in pus	140
"	"	tubercle bacilli	180
"	"	syphilis	1148
"	of	water, chemical and bacteriological	103
"	"	water, bacteriological	232
"	"	milk, market	125
"	"	milk, submitted for chemical only	0
"	"	milk, submitted for microscopic only	0
"	"	foods	28
"	"	drugs	2
"	for	courts, autopsies	3
"	"	courts, miscellaneous	19
Autopsies to complete death returns			1
Miscellaneous examinations			46
Pneumonia (sputum for typing)			0

The director of the Communicable Disease Division reports 43 cases of gonorrhea and 58 cases of syphilis.

The director of the Crippled Children's Division reports a total of 240 visits to patients during the month. Seven patients were admitted to hospitals during the month and 4 were discharged. There were 9 patients in convalescent homes at the beginning of the month; 3 patients were admitted and 3 were discharged during the month. Eighty-three pieces of apparatus were fitted during September, 40 orthopedic corrections were made to shoes, and 16 pieces of apparatus were altered or repaired. The vocational worker of this division reports sales for the month of $371.89.

The Tuberculosis Division reports that during the quarter ending September 30, 395 persons were x-rayed and 136 were tested with tuberculin. Clinic services have been offered to all colleges in the State. A simple article on tuberculosis has been prepared and will be distributed dur-

ing the sale of Christmas seals. A new health game has been prepared for schools.

The Public Health Nursing Division has opened a new unit in the town of Orwell, offering a generalized service to the towns of Shoreham, Orwell, Benson, West Haven, Sudbury and Hubbardton.

The director of the Maternal and Child Health Division attended several conferences during the month at which was discussed the development of services in connection with his work. Eight hundred and forty-six notifications of birth registration, 235 baby booklets and 700 diphtheria consent cards were sent out during the month.

The dental adviser gave several talks and spent considerable time in the construction of a model of a Vermont Health Center, which was used as a part of a display for the Vermont State Medical Society and which was later displayed at the American Dental Association meeting in St. Louis.

MISCELLANY

MAINE NEWS

Maine Medical Association

The annual fall clinical session of the Maine Medical Association was held at Lewiston on November 3 and 4.

The first day's program was held at St. Mary's Hospital and consisted of surgical clinics and a clinicopathological conference in the morning and a variety of case reports and case presentations in the afternoon.

The meetings on the second day were at the Central Maine General Hospital. There were surgical clinics and short case presentations in the morning, and a number of short case reports and papers in the afternoon. Following dinner at the DeWitt Hotel, Dr. Dennett L. Richardson, superintendent of the Charles V. Chapin Hospital, Providence, Rhode Island, spoke on "Contagious-Disease Hospitals."

Notes

Dr. Leon Babalian, a graduate of the Medical School of the University of Paris, assistant at St. Louis Hospital of Paris and a member of the Society of Dermatology and Syphilography of France, has become an associate of Dr. B. B. Foster, of Portland.

On October 20, Dr. Walter Bauer, of Boston, addressed the monthly meeting of the Kennebec County Medical Society at the Fairfield Sanatorium on "Arthritis."

Death

ABBOTT — Edville G. Abbott, M.D., died at his home in Portland, August 27, from a heart attack. He was in his sixty-eighth year. He had been in semi-retirement for the last three years.

A native of Hancock, Dr. Abbott was born November 6, 1870, the son of Alonzo and Maria Abbott. He attended the Eastern Maine Conference Seminary at Bucksport, being graduated in 1889. He then entered the granite business with his father, but five years later entered the medical school at Bowdoin College and received his degree in 1898. He continued his studies in Europe, spending a year at the Frederick Wilhelm University, Berlin, and shorter periods in London, Paris and Vienna.

On the completion of his medical studies he established himself in Portland. He was a moving force behind the establishment of the Children's Hospital there and became its first surgeon-in-chief, serving in this capacity for twenty-eight years. For ten years, from 1926-1936, he also served as chief orthopedic surgeon at the Maine General Hospital, Portland, and as consulting surgeon at the Sisters' Hospital in Lewiston, the Webber Hospital, Biddeford, and the Knox County, Augusta City and Rumford hospitals.

Dr. Abbott achieved world-wide renown through his research and skill in the prevention and cure of bone deformities, especially in children.

He had been honored by many colleges and societies. Bowdoin College gave him several honorary degrees: an A.B. in 1906, an A.M. pro merito in 1908 and an Sc.D. in 1914. Colby College honored him with an Sc.D. in 1925, and the University of Maine with an LL.D. last June. He was elected to numerous distinguished medical societies both in America and Europe.

He was a member of the Maine Medical Association, a fellow of the American College of Surgeons and a member of the National Institute of Social Sciences and of the American Academy of Political and Social Sciences. He had written a number of medical treatises.

All things considered Dr. Abbott was the foremost medical man in Maine in his day and generation.

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR SEPTEMBER, 1938

DISEASES	SEPT. 1938	SEPT. 1937	FIVE-YEAR AVERAGE*
Anterior poliomyelitis	4	141	156
Chickenpox	82	67	84
Diphtheria	10	7	25
Dog bite	832	918	781
Dysentery, bacillary	29	9	13
German measles	18	22	26
Gonorrhea	529	510	568
Lobar pneumonia	121	107	107
Measles	138	45	65
Meningococcus meningitis	5	2	4
Mumps	144	100	132
Paratyphoid B fever	16	26	6
Scarlet fever	140	168	228
Syphilis	516	427	400
Tuberculosis, pulmonary	241	280	252
Tuberculosis, other forms	19	23	30
Typhoid fever	14	16	18
Undulant fever	0	3	2
Whooping cough	339	585	483

*Based on figures for preceding five years.

RARE DISEASES

Anterior poliomyelitis was reported from: Agawam, 1; Chicopee, 1; Quincy, 1; Whitman, 1; total, 4.

Diphtheria was reported from: Fall River, 1; Lawrence, 1; Lexington, 1; Methuen, 1; Palmer, 1; Peabody, 1; Taunton, 1; Tewksbury, 1; Worcester, 2; total, 10.

Dysentery, bacillary, was reported from: Belmont, 1; Boston, 1; Danvers, 22; Lowell, 4; Lynn, 1; total, 29.

Encephalitis lethargica was reported from: Abington, 1; Brockton, 2; Canton, 1; Hingham, 1; Leominster, 1; Marshfield, 1; Quincy, 1; Waltham, 2; Wrentham, 1; total, 11.

Malaria was reported from: Brockton, 1; Cambridge, 1; Worcester, 1; total, 3.

Meningococcus meningitis was reported from: Brockton, 1; Chicopee, 1; Franklin, 1; Harwich, 1; Westfield, 1; total, 5.

Paratyphoid B fever was reported from: Arlington, 2; Cambridge, 1; Lowell, 1; Malden, 1; Norton, 4; Rehoboth, 6; West Bridgewater, 1; total, 16.

Pellagra was reported from: Boston, 1; total, 1.

Septic sore throat was reported from: Boston, 1; Malden, 1; Newton, 1; Quincy, 1; Sturbridge, 1; Winchester, 1; total, 6.

Tetanus was reported from: Boston, 1; Lexington, 1; Middleboro, 1; Somerville, 1; Watertown, 1; Worcester, 1; total, 6.

Trachoma was reported from: Chelsea, 1; Saugus, 2; Watertown, 1; Worcester, 1; total, 5.

Trichinosis was reported from: Boston, 1; total, 1.

Typhoid fever was reported from: Boston, 1; Cambridge, 1; Dedham, 1; Fall River, 1; Lynn, 1; Marlboro, 1; Medford, 1; New Bedford, 2; North Adams, 1; Quincy, 2; Somerville, 1; Woburn, 1; total, 14.

Anterior poliomyelitis showed record low incidence for the second consecutive month.

Lobar pneumonia, meningococcus meningitis, measles, and mumps were reported above the five-year average.

Scarlet fever was reported at a record low figure.

Diphtheria, pulmonary tuberculosis, whooping cough, German measles, and chickenpox were reported below the five-year average.

Tuberculosis (other forms) showed record low incidence.

Typhoid fever continued to show low incidence.

Animal rabies showed record low incidence for the second consecutive month. New foci were noted in Saugus and Wrentham.

NOTE

Dr. Edgar C. Yerbury has recently been appointed director of the Division of Mental Hygiene, Massachusetts Department of Mental Health. Dr. Yerbury is a graduate of New York University and of Boston University School of Medicine. He served as physician at the Westboro State Hospital from 1921 until 1928 and as assistant superintendent of the Danvers State Hospital from 1928 until now.

CORRESPONDENCE

UNETHICAL ADVERTISING

To the Editor: The Committee on Ethics and Discipline has been informed that an organization calling itself the Progressive Automobile Owners Association, Incorporated, of 73 Tremont Street, Boston, Massachusetts, is circularizing certain members of the medical profession regarding a booklet which is supplied to its subscribers. It states that in this booklet names of physicians are furnished for the guidance of subscribers.

A warning is issued to all fellows of the Society that the publication of such a list does not have the approval of the Committee on Ethics and Discipline.

ALEXANDER S. BEGG, M.D., *Secretary,*
Massachusetts Medical Society.

REPORT OF MEETING

NEW ENGLAND OTOLOGICAL AND LARYNGOLOGICAL SOCIETY

A meeting of the New England Otological and Laryngological Society was held April 13 at the Massachusetts Eye and Ear Infirmary, Boston. Dr. William Goodell, the president, presided. The following papers were presented:

REPORT OF SEVERAL INTERESTING BRONCHOSCOPIES. Dr. Chester R. Mills, Boston.

Case 1. A sixteen-year-old girl gave a history of having inhaled one or more common pins. An anteroposterior x-ray film showed one pin in the hypopharynx, another apparently in the chest at the level of the second and third ribs. The first was readily removed, the other could not be found either in the lung or in the esophagus. Study with the biplane fluoroscope finally revealed that what was at first thought to be a pin in the chest was a broken hypodermic needle in the soft tissues of the shoulder. Past history showed that the patient had several years previously been given a hypodermic injection which had caused her to jump.

Case 2. The chief complaints of a fifty-four-year-old man were dyspnea on exertion and after eating, frequent gastrointestinal upsets, eructations of gas, increasing constipation and precordial pressure. These signs had been increasing during the past five years. There was no loss of weight. Physical signs in the chest were consistent with compression of the lungs, especially on the right side. A preliminary diagnosis of extrabronchial new growth was made. X-rays showed a mass in the right chest, with the heart pushed to the left. Bronchoscopy was done under local anesthesia. There was complete blocking of the right main bronchus due to extrabronchial compression. Lipiodol injection showed several lobulated areas of air in the right pleural cavity. A barium meal and enema were given which showed a large diaphragmatic hernia on the right side, with the cecum and ascending colon in the right pleural cavity.

Case 3. A boy of ten was referred from the medical service because of a persistent cough, failure to gain weight, dyspnea and precordial pain. He gave a history of having been struck by a truck when he was five. He had several fractured ribs and a collapsed lung at that time. He spat up some blood and was in the hospital ten days. Since then he had been in the wards of the Boston City Hospital in 1934, 1936 and 1937, because of one or more of the above symptoms. Physical examination had always shown atelectasis of the left lower lobe. There had never been any evidence of tuberculosis. Two gastrointestinal x-ray series ruled out diaphragmatic hernia, although the signs were very suspicious. His last admission to the hospital was in December, 1937. Bronchoscopy showed the left main bronchus to be completely obstructed, with displacement of the trachea to the left. Lipiodol was instilled through the Stitt catheter. The catheter passed into the left main bronchus but the lipiodol failed to flow into the atelectatic lung. The right lung was readily outlined. The case is now being studied to decide whether lobectomy should be done.

RELATION OF PITUITARY DYSFUNCTION TO FRONTAL OSTEOMYELITIS AND PETROSITIS. Drs. Leighton F. Johnson and George Levene, Boston.

Following the work of Drs. Mortimer, Levene and Rowe at the Evans Memorial Laboratory on the typing of crania from the standpoint of pituitary dysfunction, the authors studied the incidence of osteomyelitis of the skull and petrositis through the medium of endocrinology. They found that cranial osteomyelitis of sinus origin and petrositis usually occur only in skulls showing hyperactivity of the anterior pituitary lobe. Such skulls are characterized by spongy, rarefied, vascular bone and hyperpneumatization of the sinuses, mastoids and petrous pyramids.

ATELECTASIS OF THE NEWBORN. Dr. George O. Cummings, Portland, Maine.

The author's case was that of an infant showing respiratory difficulty at the time of delivery by cesarean section. The baby never cried vigorously, and at birth a considerable amount of fluid had been drained from its pharynx by posture and aspiration. It had been given respiratory stimulants and subcutaneous saline and had at times been placed in an infant-size Drinker respirator. A chest x-ray showed atelectasis of the right upper lobe. This was obviously not due to obstetric difficulties, nor had the mother been given an excessive amount of preoperative medication. After having duly considered the usual causes of atelectasis and the fact that even massive postoperative collapse almost invariably clears in time with the shifting of the patient's position, it seemed best to let the child alone. Direct laryngoscopy with aspiration was not done because of the shock it might cause. Seventeen hours after delivery the baby died of an acute respiratory failure that suggested the sudden release of a mucous plug, with a complete blocking of the trachea.

After having discussed the various old and new methods of resuscitation, the author suggested that the bronchoscopists of the various hospital staffs should instruct residents or attending obstetricians in the use of the laryngoscope in the newborn.

USE OF PHYSICAL THERAPY IN OTOLARYNGOLOGY. Dr. John L. Myers, Kansas City, Missouri (by invitation).

This paper appeared in full in the 1937 volume of the *Transactions of the American Academy of Ophthalmology and Otolaryngology*. The author discusses the physical and therapeutic properties of the spectrum of radiation as well as the agents for the production of the various regions of this spectrum, from the humble hot-water bottle to the most complex electrical apparatus. Tumors are classified as to their response to radiation. The electrocoagulation of tonsils is put in its proper place. Sun worshippers are told that sunlight is only one of many vitalizing factors necessary to prevent disease and to maintain health. A bibliography of twenty-six titles is added. The author's conclusions are as follows:

1. Progress is being made in the therapeutic use of radium and the roentgen ray.

2. The action of light — ultraviolet, infrared and luminous rays — has not been scientifically established, though evidence enough has been given to show that through further research it may prove to be a potent therapeutic agent.

3. The medical and surgical use of heat produced by the infrared rays and the Hertzian waves is of value in combating disease.

4. Much more research is necessary before the full therapeutic value of energy in the spectrum is known.

5. There must be closer co-operation between the group work among physicians in order to secure optimum therapeutic results.

6. We must "try all things, prove all things, hold fast to that which is good."

NOTICES

THE BOSTON DISPENSARY

A luncheon meeting of the clinical staff of the Boston Dispensary will be held on Tuesday, November 29, in the auditorium on the second floor of the Dispensary building, at 12 o'clock noon.

The program, under the auspices of the gynecological department, will begin at 12:30 p. m.

PROGRAM

Cervical Biopsy. Dr. Louis E. Phaneuf.
Use of Pessaries. Dr. Herbert W. Grant.
Retained Fetal Bones in Uterus. Dr. Maurice O. Belson.
Aftercare of the Obstetric Patient from a Gynecological Standpoint. Dr. Alonzo K. Paine.

An invitation is extended to any who are interested in the subject. Luncheon to non-members, 35c.

ROBERT W. BUCK, M.D., *President*,
ALLEN R. BARROW, M.D., *Secretary*.

BOSTON DISPENSARY

25 Bennet Street, Boston
Lecture Hall, Second Floor, 9-10 a. m.

MEDICAL CONFERENCE PROGRAM, DECEMBER, 1938

Thursday, December 1 — Relation of Age to Immunity, Particularly in Respiratory Diseases. Dr. C. F. McKhann.
Friday, December 2 — Acute Intestinal Obstruction. Dr. Arnold Starr.
Saturday, December 3 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Tuesday, December 6 — Diagnosis and Treatment of Acute Pancreatic Disease. Dr. J. H. Pratt.
Wednesday, December 7 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Thursday, December 8 — Medical-Social Case Presentations of Social Service Department. Dr. I. Olef and members of the Social Service Department.
Friday, December 9 — Iodine in Relation to Thyroid Disease. Dr. W. T. Salter.
Saturday, December 10 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Tuesday, December 13 — Diagnosis of Certain Hip Conditions. Dr. J. D. Adams.
Wednesday, December 14 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Thursday, December 15 — The Use of Sulfanilamide. Dr. John Ferry.
Friday, December 16 — Bence-Jones Proteinuria. Dr. Bernard Jacobson.
Saturday, December 17 — Hospital Case Presentation. Dr. S. J. Thannhauser.

Morning medical conferences will be resumed on Tuesday, January 3, 1939.

SUFFOLK DISTRICT MEDICAL SOCIETY

There will be a meeting of the Suffolk District Medical Society on Wednesday, November 30, at 8:15 p. m., at the Boston Medical Library, 8 Fenway, Boston.

PROGRAM

Pulmonary Emboli. Dr. Aubrey O. Hampton and Dr. Benjamin Castleman.
In Vitro Dissolution of Kidney Stones. Dr. Fuller Albright.
Pre-sacral Neurectomy for Dysmenorrhea. Dr. Joe V. Meigs.
Carcinoma of the Lip. Dr. Grantley W. Taylor.
Renal Cortical Tumors. Dr. Oliver Cope.
Electroencephalography. Dr. Robert S. Schwab.
Pyelitis. Dr. Harold L. Higgins.
Recent Observations on Chronic Benzol Poisoning (Industrial). Dr. Francis T. Hunter.

Motion Picture: "New Method of Arthroplasty of the Hip." Dr. Marius N. Smith-Petersen.

JOHN P. MONKS, M.D., *Secretary.*

GREATER BOSTON MEDICAL SOCIETY

The next meeting of the Greater Boston Medical Society will be held at the Beth Israel Hospital Auditorium on Tuesday, December 6, at 8:15 p. m.

Dr. Max M. Peet, professor of surgery, University of Michigan Medical School, will speak on "The Surgical Treatment of Hypertension." This will be followed by a discussion by Dr. David Ayman, Dr. Reginald H. Smithwick and Dr. James C. White.

L. M. FREEDMAN, M.D., *President,*
D. B. STEARNS, M.D., *Secretary.*

FAULKNER HOSPITAL CLINICOPATHOLOGICAL CONFERENCE

The regular monthly clinicopathological conference of the Faulkner Hospital for its staff and any other interested members of the medical profession will be held on Thursday, December 1, at 5:00 p. m.

Cases will be discussed by Dr. Channing Frothingham and Dr. Everett E. O'Neil.

CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS, UNDER THE PROVISIONS OF THE SOCIAL SECURITY ACT

CLINIC	DATE	ORTHOPEDIC CONSULTANT
Lowell	December 2	Albert H. Brewster
Salem	December 5	Harold C. Bean
Haverhill	December 7	Arthur T. Legg
Brockton	December 8	George W. Van Gorder
Gardner	December 13	Mark H. Rogers
Worcester	December 16	John W. O'Meara
Pittsfield	December 19	Francis A. Slowick
Springfield	December 21	Garry deN. Hough, Jr.
Hyannis	December 27	Paul L. Norton
Fall River	December 28	Eugene A. McCarthy

NORFOLK DISTRICT MEDICAL SOCIETY

The next meeting of the Norfolk District Medical Society will be held in the Hotel Somerset, Boston, Tuesday, November 29, at 8:30 p. m. Tel. Com. 2700.

PROGRAM

Communication: Clinical Immunology of Staphylococcus Infections. Dr. Champ Lyons.
Discussion.
Collation.

FRANK S. CRUICKSHANK, M.D., *Secretary.*

PAN-PACIFIC SURGICAL ASSOCIATION

The third congress of the Pan-Pacific Surgical Association is to be held in Honolulu, September 15 to 28, 1939. An invitation is extended to all surgeons of the state medical societies to meet, in Honolulu, outstanding men from countries of the Pacific area, including Australia, New Zealand, China, Japan, Java, Canada and the United States, for an interchange of surgical thought and for the purpose of bringing about better understanding through personal contact.

There will be sections on fractures and orthopedics, general surgery, gynecology, neurosurgery, ophthalmology, otolaryngology, roentgenology, plastic surgery, thoracic surgery and neurology, all in charge of outstanding men as chairmen for the United States and equally prominent men as chairmen for the Australasian section. The congress affords not only participation in interesting scientific papers, but a most enjoyable vacation in the "Paradise of the Pacific."

Communications for information should be directed to George W. Swift, M.D., 902 Boren Avenue, Seattle, past president of the Association; Frederick L. Reichert, M.D., Stanford University Hospital, San Francisco, program chairman for the United States; Howard Updegraff, M.D., 6777 Hollywood Boulevard, Los Angeles, program vice-chairman; or Forrest J. Pinkerton, M.D., secretary-treasurer of the Association, Young Building, Honolulu, Hawaii.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, NOVEMBER 28

TUESDAY, NOVEMBER 29

*9-10 a. m. Boston Dispensary. The Value of Urethrography in the Diagnosis and Treatment of Lesions of the Lower Genito-Urinary Tract. Dr. M. L. Brodny.

9:30 a. m. Massachusetts General Hospital, Thoracic clinic.

*10 a. m. to 12:30 p. m. Tumor clinic. Boston Dispensary.

*12 m. Boston Dispensary. Luncheon meeting of the clinical staff.

8:30 p. m. Norfolk District Medical Society. Hotel Somerset, Boston.

WEDNESDAY, NOVEMBER 30

8 a. m. Massachusetts General Hospital. Grand rounds, orthopedic department.

*9-10 a. m. Boston Dispensary. Hospital case presentation. Dr. S. J. Thannhauser.

*12 m. Clinicopathological conference. Children's Hospital amphitheater.

8:15 p. m. Suffolk District Medical Society. Boston Medical Library.

THURSDAY, DECEMBER 1

8:30-9:30 a. m. Exchange visit, Surgical and Orthopedic Staffs of the Peter Bent Brigham and Children's hospitals, held this week at the Peter Bent Brigham Hospital.

9 a. m. Massachusetts General Hospital. Surgical grand rounds.

9 a. m. Massachusetts General Hospital. Neurological staff conference.

*9-10 a. m. Boston Dispensary. Relation of Age to Immunity, Particularly in Respiratory Diseases. Dr. C. F. McKhann.

*10:30 a. m. Psychiatric Rounds. Boston City Hospital. Rounds will start at Room 916 on the ninth floor of the Medical Building.

12 m. Massachusetts General Hospital. Clinicopathological conference.

5 p. m. Faulkner Hospital. Clinicopathological conference.

FRIDAY, DECEMBER 2

*9-10 a. m. Boston Dispensary. Acute Intestinal Obstruction. Dr. Arnold Starr.

10 a. m. Massachusetts General Hospital. Fracture rounds.

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

12 m. Clinical meeting of the Children's Medical Service, Massachusetts General Hospital, Ether Dome.

SATURDAY, DECEMBER 3

*9-10 a. m. Boston Dispensary. Hospital case presentation. Dr. S. J. Thannhauser.

*10 a. m. - 12 m. Staff rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

*Open to the medical profession.

NOVEMBER 29 — Boston Dispensary. Luncheon meeting of clinical staff. Page 862.

DECEMBER 1 — Faulkner Hospital. Clinicopathological conference. Notice above.

DECEMBER 1-17 — Boston Dispensary, Medical Conference Program. Page 862.

DECEMBER 2-28 — Clinics for Crippled Children in Massachusetts, Under the Provisions of the Social Security Act. Notice above.

DECEMBER 6 — Greater Boston Medical Society. Notice above.

DECEMBER 6 — Lawrence Cancer Clinic. Page 814, issue of November 17.

DECEMBER 8 — Pentucket Association of Physicians, 8:30 p. m., Hotel Bartlett, 95 Main Street, Haverhill.

JANUARY 12, 1939 — Peter Bent Brigham Hospital. Clinic conducted by Dr. Christian. Page 633, issue of October 20.

FEBRUARY 4, MAY 15 and 16, 1939 — American Board of Obstetrics and Gynecology. Page 451, issue of September 22. *(Application for admission to Group A examinations must be on file in the Secretary's office by March 15, 1939, instead of April 1 as previously stated.)*

MARCH 27-31 — American College of Physicians. Page 36, issue of July 7.

MAY 7-15 — International Congress of Military Medicine and Pharmacy. Page 501, issue of September 29.

MAY 15-19 — American Medical Association, St. Louis, Missouri.

JUNE 6, 7, 8 — Massachusetts Medical Society. Worcester.

SEPTEMBER — Boston Psychoanalytic Institute. Page 450, issue of September 22.

SEPTEMBER 15-28 — Pan-Pacific Surgical Association. Page 863.

DISTRICT MEDICAL SOCIETIES

ESSEX SOUTH

DECEMBER 7 — Salem Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speakers: Dr. Walter G. Phippen and Dr. Channing Frothingham. Subject: Economic Problems.

JANUARY 4 — Danvers State Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. Kenneth J. Tillotson. Subject: The Psychiatrist's Viewpoint in Delinquency.

FEBRUARY 8 — Essex Sanatorium, Middleton. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. Edward Churchill. Subject: Surgical Treatment of Pulmonary Suppuration.

MARCH 1 — Lynn Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. John Rock. Subject: Endocrinology.

APRIL 5 — Addison Gilbert Hospital, Gloucester. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. Ethan Allan Brown. Subject: Allergy.

MAY 10 — Annual meeting. Salem Country Club, Peabody.

NORFOLK

NOVEMBER 29 — Page 863.

SUFFOLK

NOVEMBER 30 — Page 862.

JANUARY 25, 1939 — Symposium on Diabetes. Dr. Elliott P. Joslin and associates, Boston Medical Library, 8:15 p. m.

MARCH 29 — Joint meeting with New England Pediatric Society, Boston Medical Library, 8:15 p. m. Program and speakers to be announced.

APRIL 26 — Annual meeting in conjunction with Boston Medical Library, at 8:15 p. m. Election of officers. Program and speakers to be announced.

BOOK REVIEWS

The Biology of Arteriosclerosis. M. C. Winternitz; R. M. Thomas and P. M. LeCompte. 142 pp. Springfield, Illinois and Baltimore: Charles C Thomas, 1938. $4.00.

With a prediction that the next great campaign for health will be directed against arteriosclerosis, and with a conviction that the extensive investigations of the past thirty years have not clarified materially our understanding of the cause of this condition, Winternitz and his colleagues have ventured a new method of attack on this important problem. In an exuberantly illustrated monograph the authors have incorporated thoughts on this and other subjects, criticisms of current and past theories concerning the nature and cause of arteriosclerosis, the results of observations on human and animal material, and finally a discussion replete with theoretical considerations. "Old tools have proved indispensable in this study of the blood vessels and their diseases," state the authors, and they are to be congratulated on their demonstration of high technical skill with methods to which they have brought new life.

Their conception of the biology of arteriosclerosis is based on their designation of the artery "as a vascular or potentially vascular organ," subject to the same pathologic processes as other tissues. By means of India-ink injections and clearing methods, rich vascular networks were demonstrated within and around arteriosclerotic plaques (a not surprising finding) and in the blood vessels of several animal species. Relatively few vessels were demonstrated in the normal arteries of young individuals. Numerous vasa were found in the veins, and in all coats of the pulmonary artery.

The relation of these vascular networks to arteriosclerosis is then pointed out. As one bit of evidence, "circumstantial, perhaps, but very striking," the authors call attention to their observation that hemorrhages abound in just those regions which are the sites of origin of vasa. Extravasated blood is regarded as the main source of atheromatous material. Some relation, therefore, among three factors — vascularity, hemorrhage and sclerosis — seems likely. The sequence of events in arteriosclerosis is considered to be: damage to vasa vasorum, which permits hemorrhage or exudation, with consequent productive changes leading to the formation of new vessels and connective tissue. Indirect evidence is obtained from a study of "adaptive changes," which include the obliteration of the fetal vessels at birth, the changes in the uterine artery with pregnancy, and the obliteration of large pulmonary vessels which traverse tuberculous cavities.

The reader is informed only in general terms, which border on vagueness surprising in a scientific work, concerning the nature of deleterious agents and the causes of hemorrhage and exudation which take place from actual vessels or which might take place from "potential vessels." The critical reader will object to the use of unphysiologic pressures (in some instances as high as 1500 mm. of mercury) in the demonstration of a vessel pattern which forms the basis of the authors' conception of arteriosclerosis. He will deplore the frequent use of uncertain phrases such as "suggestive evidence," "strong presumptive evidence of their potential presence" and "tendency to exudation" in situations where proof logically might be expected.

The character of the illustrations, many in color, deserves more than passing comment. The authors have been less fortunate in the number of facts gleaned from the amount of labor evident in both the illustrations and the text. The monograph is provocative without question. The reviewer, however, will await with interest the publication by the authors of proof for their many stimulating hypotheses. That the authors themselves regard their task as unfinished, may be deduced from their concluding sentence (italics the reviewer's): "But surely an approach to the problem which is based on recognition of the artery as a vascular or a *potentially* vascular organ, and therefore subject to the same pathological processes to which other tissues are subject, *may prove* more fruitful than one in which the lesions are regarded as primarily 'degenerative' or as the inevitable concomitants of age."

A Textbook of Nursing Practice. Hester K. Frederick and Ethel Northam. Second edition (revised). 418 pp. New York: The Macmillan Co., 1938. $3.00.

These authors, graduates of the Johns Hopkins Hospital School of Nursing, with subsequent important executive and teaching positions, have contributed a standard textbook of nursing practice.

Beginning with an explanation of the relation of the art and practice of nursing to hospital administrations, the reader is conducted through the various steps in the process of teaching nurses to take their proper positions as assistants to doctors and caring for patients. Without attempting to make doctors of the nurses, all details of the nurses' problems in routine practice are set forth clearly, as well as sufficient information about medical problems which nurses ought to have.

Books of the quality of this one should be made available to pupil nurses and perhaps in some places to the instructors on the teaching staff.